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The Synopsis of FORENSIC MEDICINE and Toxicology

29th Edition

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JAYPEE

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By

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The Synopsis of Forensic Medicine and Toxicology

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Preface

"To study disease without books is to sail an uncharted sea, while to study books alone is not to go to sea at all".

—William Osler

"Some books are to be tasted, others to be swallowed and some few to be chewed and digested."

—Bacon

This is condensed version of the treatise published under the title: "The Essentials of Forensic Medicine and Toxicology" thirty-fourth edition, 2017. It is designed to provide a brief and essentially practical guide to current teaching in Forensic Medicine with particular reference to India. The subject matter has been dealt with concisely, which is easy to grasp and simplified in presentation, and wherever necessary illustrations, tables and points have been inserted to help the students. The aim is to provide reasonable coverage of the subject as a whole. An attempt has been made to maintain the practical character of the book. Over and above, the book has been entirely revised and special additions and alterations have also been made and the overall text brought up-to-date.

It is intended primarily to meet the needs of the undergraduate medical students already overburdened with a heavy curriculum, to have a clear grasp of the subject and for examination purpose. It is a complete textbook, which provides a reasonable and up-to-date coverage of the subject as a whole.

The undergraduate medical students may omit the text printed in smaller type, except tables. I have consulted various textbooks and periodicals in the preparation of this book, to the authors of which, I am grateful. The opinions expressed in the text are entirely mine. I am also grateful to many friends and professional colleagues for their help and advice and to all those whose comments and criticisms have helped to keep revisions of this book accurate and up-to-date, which is hereby acknowledged wholeheartedly. It is requested that the discrepancies, if any noticed, may please be intimated to me, so that it may be taken care of in the next edition. Suggestions for improvement are welcome.

The physical pathology of trauma alters little over the years, but academic and practical approaches to the subject do evolve. Accordingly, I have attempted to introduce new ideas. It is said that the proof of the pudding lies in the eating. The popularity of the previous editions of this title prompted the publication of this edition. It is hoped that this edition will be warmly welcomed just like the previous editions of this treatise.

I am grateful to all those associated with M/s Jaypee Brothers Medical Publishers (P) Ltd, New Delhi, India, and particularly Shri Jitendar P Vij (Group Chairman) and Mr Ankit Vij (Group President).

K.S.N. REDDY

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CHAPTER

1 Introduction

Forensic or legal medicine (forensic = of or used in Courts of law) **deals with the application of medical and paramedical knowledge to aid in the administration of justice**. It is used by the legal authorities for the solution of legal problems. Some examples are applying the medical knowledge in deciding cases of injuries, murder, suicide, accidents, asphyxial deaths, sexual offences, poisoning, etc. **In short, it deals with medical aspects of law**.

Medical jurisprudence (*juris* = law; *prudentia* = knowledge) **deals with legal responsibilities of the physician with particular reference to those arising from physician–patient relationship**, such as medical negligence cases, consent, rights and duties of doctors, serious professional misconduct, medical ethics, etc. **In short, it deals with legal aspects of practice of medicine.**

Medical ethics deals with the moral principles which guide members of the medical profession in their dealings with each other, their patients and the State.

Medical etiquette deals with the conventional laws of courtesy observed between members of the medical profession.

Forensic medicine deals almost entirely with crimes against human beings, in which medical examination and evidence are required. Forensic medicine is mostly an exercise of common sense, combined with the application of knowledge and experience, already acquired in the study of other branches of medicine, surgery, obstetrics, etc. **Its aim is to find out the truth. Its particular field of activity is judicial investigation, both civil and criminal.** All medical work is of a responsible character, especially the medicolegal work, such as issuing certificates of lunacy, sickness, etc. In all cases of crime involving the person, e.g. homicide, suicide, assault, sexual offences, traffic accidents, poisoning, etc., the help of the medical practitioner is sought by the police. In all such cases, the doctor will be required to appear as an expert witness in a Court of law. In cases of sudden death, the authorities will depend mostly or completely on medical evidence in establishing the cause of death, and in case of accident to determine blame.

The Court has no special medical knowledge. It relies on his witness for an opinion and expects him to assist it with his special knowledge and experience. **Other things being equal, the better the presentation of medical evidence, the better is the administration of justice.** The pathologist must be guarded in what he says, and how he says it. The medical expert should be very careful when he is examining living people. **He should not encourage an accused person to talk about the crime with which he is charged, or about the events that led to his arrest. If, during a medical examination, an alleged accused says anything that might incriminate himself, it should be neither recorded nor reported. The medical evidence does not itself prove the case of prosecution.**

Three things are needed for success: (1) the power of observation, (2) a wide range of exact knowledge, and (3) the power of deduction. There is no substitute for basic intelligence and clinical competence. Experience, commonsense, and willingness to consider other possibilities are as essential in the practice of forensic medicine as a wide range of theoretical knowledge. **A good command of language, clear presentation, and ability in expressing a relatively firm opinion are necessary for the success of the forensic pathologist.** The forensic pathologist must be alert to where evidence should be looked for, and how it should be interpreted.

Forensic medicine is not an exact science. Unexpected results are produced due to biological variations. In every case, there is an element of uncertainty, and **absolute proof is a rarity in any**

medical problem. Doctors should bear in mind, the essential difference between probability and proof. The medical witness should not be dogmatic about his opinion. **The doctor should be ready to defend every finding and conclusion on the report on clinical and scientific grounds.** He should be aware of professional and scientific viewpoints which might differ from his, and should be familiar with the latest scientific literature in relation to the subject involved. For the purpose of illustrating and clarifying his testimony, the medical expert may employ photographs, maps, diagrams, charts, X-rays, skeletons, models, etc. when they are properly verified.

The doctor must be honest, for confidence is inspired by honesty and success depends upon confidence. A doctor should not become partisan. He must bear testimony within the limits of science. His sole obligation is to present the truth as he sees it, adding nothing, withholding nothing and distorting nothing. If evidence has been properly gathered and preserved, a mistake in interpretation may always be corrected. **The medicolegal aspects of any case must always be secondary to life-saving treatment of the patient.**

HISTORY: Law-medicine problems are found in the written records in Egypt, Sumer, Babylon, India and China dating back 4000 to 3000 B.C. A Chinese materia medica of about 3000 B.C. gives information on poisons. The Code of Hammurabi, King of Babylon (about 2200 B.C.) is the oldest known medicolegal code. Hippocrates (460 to 377 B.C.), the "Father of Western Medicine", who was born and practised in the island of Kos in Greece, discussed the lethality of wounds and contributed much to medical ethics. About 300 B.C., the Jewish laws sought the aid of medical expert in the administration of justice. Around the beginning of the Christian era, various eugenic and public health provisions were laid down in the Laws of Manu, and many sexual matters were brought under the law in India. In the sixth century A.D. the Justinian Code regulated the practice of medicine and surgery, and established the function of the medical expert in legal proceedings. The first medicolegal autopsy was done in Bologna (Italy) in 1302. In the thirteenth century, a manual was prepared to aid in the investigation of death in China. In the sixteenth century, the Penal Code of the Bishop of Bamberg, and the Caroline Code, both increased importance of legal medicine by their insistence that medical testimony was an essential part of the proof in trials. Around the end of the sixteenth century, autopsies in medicolegal cases began to be generally practised. The first book on forensic medicine was published in 1602, by an Italian physician. The greatest of all works was the "Questiones Medicolegalis" (medicolegal questions), written by Paulus Zacchias (Father of Legal Medicine and Forensic Psychiatry) in the seventeenth century, who was principal physician to Pope Innocent X and Alexander VII. This work remained an authority in medicolegal matters until the beginning of the nineteenth century. In the eighteenth century, professorships in legal medicine were founded by the State in Germany. Orfila (1787 to 1853) introduced precise chemical methods into toxicology. In 1843, the law regarding the criminal responsibility of insane persons was established in England in Mc Naghten's case.

CHAPTER

2 Legal Procedure

INDIAN PENAL CODE, 1860 (I.P.C.): It deals with substantive criminal law in India. It defines offences and prescribes punishments.

CRIMINAL PROCEDURE CODE, 1973 (Cr.P.C.): It provides the mechanism for punishment of offences against the substantive criminal law. It deals with police duties in arresting offenders, dealing with absconders, in the production of documents, etc. and in investigating offences. It provides for different class of Courts and deals with actual procedure in trials, appeals, references, revisions and transfer of criminal cases.

INDIAN EVIDENCE ACT, 1872 (I.E.A.): It deals with Law of Evidence, which applies to all judicial proceedings in any Court. It is common to both the criminal and civil procedures.

CIVIL LAW: It deals with disputes between two individuals or parties. The party bringing the action in a civil case is called "plaintiff". The accused is called 'defendant' in both criminal and civil cases.

CRIMINAL LAW: It deals with offences which are considered to be against the public interest, such as offences against the person, property, public safety, security of the State, etc. Here the State is a party represented by public prosecutor, and the accused is the other party.

INQUEST: An inquest is an enquiry or investigation into the cause of death. It is conducted in cases of suicide, homicide, accidents, suspicious (unnatural) deaths and death due to anaesthesia or during operation. Two types of inquests are held in India.

(1) POLICE INQUEST: The officer-in-charge of a police station (usually subinspector but not below the rank of head constable) conducts the inquest (S.174, Cr.P.C.). The police officer making the inquest is known as Investigating Officer (I.O.). When the officer-in-charge of a police station receives information that a person has committed suicide, or has been killed by another or by an animal or by machinery or by an accident, deaths due to torture or ill-treatment, suspected medical negligence, or has died under circumstances raising a reasonable suspicion, that some other person has committed an offence, and unidentified or skeletonised bodies, he immediately gives intimation thereof to the nearest Executive Magistrate empowered to hold inquests, and proceeds to the place where the body of such deceased person is. There, in the presence of two or more respectable persons *(panchas),* makes an investigation **(S.175, Cr.P.C.). He prepares a report of the apparent cause of death, describing wounds, fractures, bruises, and other marks of injury found on the body,** and stating in what manner, or by what weapon or instrument, such injuries appear to have been inflicted. The inquest report *(panchanama)* is then signed by the investigating police officer and by the *panchas*. If no foul play is suspected, the dead body is handed over to the relatives for disposal. In cases of suspected foul play or doubt, the body is sent for postmortem examination together with a requisition to the authorised Government medical officer.

Private medical institutions can undertake medicolegal examination of the living including treatment, but autopsies can be conducted only with the permission of the State Government.

(2) MAGISTRATE'S INQUEST: This is conducted by a District Magistrate, Sub-divisional Magistrate, or any other Executive Magistrate, especially empowered by the State Government or the District Magistrate, such as Collector, Deputy Collector or *Tahsildar* **(Executive Magistrates).** It is done in cases of (1) death in police custody and while under police interrogation, (2) death due to police firing, (3) death in prison, reformatories, Borstal School, (4) dowry death, (5) death in a psychiatric hospital, (6) exhumation, (7) death or disappearance of any person, or rape alleged to have been committed on any woman, while such person or woman is in the custody of the police or any others custody authorised by the court.

In any case of death, a Magistrate may conduct an inquest instead of, or in addition to the police inquest (S.176, Cr.P.C.).

Other types:

(1) CORONER'S INQUEST: **This is a type of inquest done in the United Kingdom, some States in the United States of America and some other countries but not in India. Coroner conducts inquest in all unnatural and suspicious deaths. The doctor is summoned to his Court to give evidence at the inquest. Coroner's Court is a Court of enquiry, wherein Jurors are sworn in to give a true verdict according to the evidence. He has some judicial powers.**

(2) MEDICAL EXAMINER'S SYSTEM: **This is a type of inquest done in most States in the United States of America, but not in India. A medical practitioner, known as Medical Examiner, is appointed to perform the functions of Coroner. He does not have any judicial functions similar to the Coroner. As the doctor visits the scene of crime and conducts the inquest, it is superior to Coroner's and police inquest.**

JURY: **Jury is a group of responsible, educated persons of good social position called "Jurors". Trial by jury has been abolished in India.**

COURTS OF LAW

Types: (1) Civil and (2) Criminal. **The criminal Courts in India are of four types.**

(1) Supreme Court is the highest judicial tribunal, and is located in New Delhi. It has power of supervision over all Courts in India. It is purely an appellate Court in criminal cases. The law declared by it is binding on all Courts (**Article 141 of Constitution of India**).

(2) The **High Court** is usually located in the capital of every State and is highest tribunal for the State. It may try any offence and pass any sentence authorised by law (**S.28, Cr.P.C.**). Judges of the High Court are appointed by the President of India.

The Supreme Court and High Courts act as Courts of appeal only in criminal cases and do not hold trial *prima facie.*

(3) The **Sessions Court** is usually located at the district headquarters. **It can only try cases which have been committed to it by a Magistrate** (**S. 193, Cr.P.C.**). **It can pass any sentence authorised by law, but a sentence of death passed by it must be confirmed by the High Court** (**S. 28(2) and S. 366, Cr.P.C.**). **District Court deals with civil cases.** An **Assistant Sessions Court** can pass sentence of imprisonment up to 10 years and unlimited fine (**S. 28(3), Cr.P.C.**).

Table (2-1). Powers of Magistrates (S.29, Cr.P.C.).

Class of Magistrate	Imprisonment	Fine
Chief Judicial Magistrate	Up to 7 years	Unlimited
I Class Judicial Magistrate	Up to 3 years	10,000 Rupees
II Class Judicial Magistrate	Up to 1 year	5,000 Rupees

(4) Magistrates are of three types:

(1) Chief Judicial Magistrate.

(2) First Class Judicial Magistrate.

(3) Second Class Judicial Magistrate.

In metropolitan cities with a population of more than one million, the Chief Judicial Magistrate and First Class Judicial Magistrate are designated as Chief Metropolitan Magistrate and Metropolitan Magistrate, respectively (**Table 2-1**).

Sessions judges and magistrates are appointed by the High Court.

When a person is convicted at one trial for two or more offences, twice the amount of punishment, which a Magistrate is authorised, can be passed (**S.31, Cr.P.C.**).

Juvenile Courts: They try offences committed by Juveniles (below the age of 18 years) (**S.27, Cr.P.C.**). Juvenile courts are usually presided by a first class woman magistrate.

Any sentence awarded by a Court may be enhanced or decreased by a higher Court.

The President of India has the power to grant pardons, remissions of punishments, and commutation of death sentence. State government has power to suspend the execution of any sentence or to remit whole or any part of the punishment passed by a court of law (**S. 432 and 433, Cr.P.C.**).

COGNISABLE OFFENCE: **It is an offence in which a police officer can arrest a person without warrant from the Magistrate, e.g. rape, murder, dowry death, rash or negligent act, etc.** (**S.2(c), Cr.P.C.**). In such offences, the individual is sent by the police to the doctor for medical examination. In **non-cognisable offences**, the injured person may go

direct to the doctor, or he may file an affidavit in the Court of a Magistrate who will send him to the doctor for examination (S.41, Cr.P.C.).

Punishments: The sentences authorised by law are: (1) death, (2) imprisonment for life, (3) imprisonment: (a) rigorous, i.e. with hard labour, (b) simple, (4) forfeiture of property, (5) fine (S. 53, I.P.C.).

Capital Punishment (Death Penalty): The various methods of carrying out death sentence are: hanging, electrocution, shooting, gas chamber, lethal injection [sodium thiopental (unconsciousness), pancuronium bromide (paralytic, stops breathing) and potassium chloride (stops heart)], garrotting and guillotine (a method of decapitation).

SUBPOENA OR SUMMONS: Sections 61 to 69 of Cr.P.C. deal with summons. **Subpoena** (sub = under; poena = penalty) **is a document compelling the attendance of a witness in a Court of law under penalty, on a particular day, time and place for the purpose of giving evidence. It may also require him to bring with him any books, documents or other things under his control, that he is bound by law to produce in evidence.** It is issued by the Court in writing, in duplicate, signed by the presiding officer of the Court and bears the seal of the Court. It is served on the witness by a police officer, or other public servant, by delivering to him one of the copies of the summons. The person should sign a receipt on the back of the other copy. If the person summoned is in the active service of the Government, the Court issuing the summons shall usually send it in duplicate to the head of the office in which such person is employed, and such head shall cause the summons to be served on such person, and shall return it to the Court under his signature with the endorsement required by that section (S.66, Cr.P.C.). A summons must be obeyed, and the witness should produce documents if asked for (S.91, Cr.P.C.). The witness will be excused from attending the Court, if he has a valid and urgent reason. If the witness fails to attend the Court: (1) In a civil case, he will be liable to pay damages. (2) In a criminal case, to fine or issuing a warrant for arrest (S.172 to 174, I.P.C., and S.87, Cr.P.C.).

Priority: Criminal Courts have priority over Civil Courts. If a witness is summoned by two Courts on the same day, one of which is criminal and other civil, he should attend the Criminal Court and inform the Civil Court of his inability to attend, giving his reasons. **Higher Courts have priority over the lower.** If summoned to two Courts on the same day, he must first attend the higher Court. If he is summoned from two Courts of same status, he must attend the Court from where he received the summons first, informing the other Court about it. He can attend the second Court after finishing his evidence in the first Court.

CONDUCT MONEY: It is the fee paid to a witness in civil cases at the time of serving the summons, to meet the expenses for attending a Court. If the fee is not paid, or if he feels that the amount is less, the doctor can bring this fact to the notice of the Judge before giving evidence in the Court. The Judge will decide the amount to be paid.

In criminal cases, no fee is paid to the witness at the time of serving the summons. He must attend the Court and give evidence; otherwise he will be charged with contempt of Court. However, in criminal cases conveyance charges and daily allowance can be claimed by the doctor according to the Government rules. **Contempt means wilful disregard or disobedience of court's order** or conduct which tends to bring the authority of the court and administration of law into disrepute.

MEDICAL EVIDENCE

Evidence means and includes: (1) all statements which the Court permits or requires to be made before it by witnesses, in relation to matters of fact under inquiry, (2) all documents produced for inspection of the Court (S.3, I.E.A.).

Types: (1) Documentary: Sections 61 to 90 of Indian Evidence Act, 1872, deal with documentary evidence. The contents of documents may be proved either by primary or by secondary evidence (S.61, I.E.A.). **Primary evidence** means the document itself produced for inspection of the Court (S.62, I.E.A.). **Secondary evidence** means and includes (a) Certified copies. (b) Copies made from the original by mechanical processes. (c) Copies made from or compared with the original. (d) Oral accounts of the contents of a document. **(2) Oral. (a) Direct:** Evidence of a fact which is actually in issue, e.g., an electric blanket that has

caused injury, a prescription, or a consent form. **(b) Indirect or circumstantial:** Evidence of a fact which is not actually in issue, but which is derived from the circumstances, e.g., in case of alleged murder of A by B at certain place on a particular day and time, the circumstantial evidence would be that C saw B with a knife on that day at that place, a few minutes before the murder.

(5) Hearsay: It is any statement made by any person other than the witness giving evidence in Court, which is presented in the Court in order to assert that the facts contained in the statement are true, e.g., A gives evidence in the witness box stating that B had informed him that he had seen C commit a crime. In such case direct evidence can be given only by B that he had seen C committing a crime.

DOCUMENTARY EVIDENCE: It is of three types.

(1) MEDICAL CERTIFICATES: They refer to ill-health, insanity, age, death, etc. They are accepted in a Court of law, only when they are issued by a qualified registered medical practitioner. In case of ill-health, the exact nature of ill-health and probable period of expected absence should be stated and the signature or left thumb impression of the patient should be taken at the bottom of the certificate. **A medical practitioner is legally bound to give a death certificate, stating the cause of death without charging fee, if a person whom he has been attending during his last illness dies (Registration of Births and Deaths Act, 1970).** Death certificate should not be issued by a doctor without inspecting the body himself, and satisfying that the person is really dead. The certificate should not be delayed, even if the doctor's fees is not paid. **The certificate should not be given, if the doctor is not sure of the cause of death, or if there is the least suspicion of foul play. In such cases, the matter should be reported to the police.**

(2) MEDICOLEGAL REPORTS: They are reports prepared by a doctor usually in criminal cases, e.g., assault, rape, murder, etc. The examination of an injured person or a dead body is made, when there is a requisition from a police officer or Magistrate. **These reports consist of two parts: (1) the facts observed on examination, and (2) the opinion drawn from the facts.** These reports will be attached to the file relating to the case, and the file is produced in the Court. The report will be open to the scrutiny of the defence lawyer. It will not be admitted as evidence, unless **the doctor attends the Court and testifies to the facts under oath.** Great care should be taken in writing the reports to avoid any loose wording or careless statement. This gives a chance to the defence lawyer to use them to his own advantage. The report should give the date, time and place of examination and the name of police officer who identified the person or the dead body. Exaggerated terms, superlatives, etc. should not be used. **The opinion should be based on the facts observed by himself, and not on information obtained from other sources. In an injury case, if it is not possible to give an opinion immediately, the person should be kept under observation,** and necessary investigations should be done before giving the report. The doctor should sign or initial at the bottom of each page, if the report exceeds one page in length. **The report should be written in duplicate using carbon papers.** The first copy is sent to the investigating officer in a sealed cover and another copy retained for future reference. Clothing, weapons, etc., sent for medical examination should be described in detail, sealed and returned to the police, after obtaining a receipt.

(3) DYING DECLARATION: It is a written or oral statement of a person, who is dying as a result of some unlawful act, relating to the material facts of cause of his death or bearing on the circumstances (S.32, I.E.A.). If there is time, Executive Magistrate should be called to record the declaration. Before recording the statement, the **doctor should certify that the person is conscious and his mental faculties are normal** ***(compos mentis).*** If the condition of the victim is serious, and there is no time to call a Magistrate, the doctor should take the declaration in the presence of two witnesses. The statement can also be recorded by the village headman, police or any other person, but its evidential value is less. **While recording the dying declaration, oath is not administered, because of the belief that the dying person tells the truth.**

Statement: The statement should be recorded in the man's own words, without any alteration of terms or phrases. Leading questions should not be put. The declarant should be permitted to give his statement **without any undue influence, outside**

prompting or assistance. If a point is not clear, question may be asked to make it clear, but the actual question and the answer received should be recorded. It should then be read over to the declarant, and his signature or thumb impression is taken. The doctor and the witness should also sign the declaration. If the statement is written by the declarant himself, it should be signed by him, the doctor and the witnesses. **The statement made must be of fact and not of opinion.** If the declaration is made in the form of an opinion or conclusion, questions should be asked by the recorder to bring out the facts that are the basis for the conclusion. While recording the statement, if the declarant becomes unconscious, the person recording it must record as much information as he has obtained and sign it. **If the dying person is unable to speak but is able to make signs in answer to questions put to him, this can be recorded and it is considered as a "verbal statement".**

Admissibility: The declaration is admissible not only against an accused who killed the declarant, but also against all other persons involved in the same incident which resulted in his death. In India, if the declarant is in a sound state of mind at the time of making the declaration, **it is admissible in Court as evidence, even if the declarant was not under expectation of death at that time.** The statement is important to identify the offender or to clear innocent persons. The declaration is sent to the Magistrate in a sealed cover. **It is produced at the trial and accepted as evidence in case of death of the victim in all criminal and civil cases, where the cause of death is under enquiry.** The person recording the declaration will have to give evidence in the Court to prove it. **If the declarant survives, the declaration is not admitted but has corroborative value, and the person is called to give oral evidence.**

DYING DEPOSITION: It is a statement of a person on oath, recorded by the Magistrate, in the presence of the accused or his lawyer, who is allowed to cross-examine the witness. <u>This procedure is not followed in India</u>.

ORAL EVIDENCE: It includes all statements which the Court permits, or which are required to be made before it by the witness, in relation to matters of fact under enquiry. All facts, except the contents of documents, may be proved by oral evidence **(S. 59, I.E.A.). In all cases, oral evidence must be direct.** It must be evidence of a person who saw, heard, or perceived it by that sense or in that manner. If it refers to an opinion or to the grounds on which the opinion is held, it must be the evidence of the person who holds that opinion on those grounds **(S. 60, I.E.A.). A deaf and mute witness may testify by signs, by writing, or through an interpreter.** If oral evidence refers to any material thing, the Court may require the production of such a thing for its inspection, e.g., a bloodstained weapon or article of clothing, a portion of eliminated poison, etc. **(S. 60, I.E.A.). Oral evidence is more important than documentary evidence, as it admits cross-examination. Documentary evidence is accepted by the Court only on oral evidence by the person concerned.**

EXCEPTIONS TO ORAL EVIDENCE: (1) Dying declaration (S. 32 and 157, I.E.A.). (2) Expert opinion expressed in a treatise (S.60, I.E.A.). (3) Evidence of a doctor recorded in a lower Court. (4) Evidence given by a witness in a previous judicial proceeding. (5) Evidence of Mint Officers. (6) Reports of certain Government scientific experts: (a) Chemical Examiner. (b) Chief Inspector of Explosives. (c) Director Fingerprint Bureau. (d) Director, Haffkine Institute, Mumbai. (e) Director, Central Forensic Science Laboratories or State Forensic Science Laboratories. (f) Serologist to the Government of India. The Court has the power to summon and examine any such expert. The prosecution and defence have also a right to demand the Court to summon and examine any such expert. (7) Public records: A record kept in a public office, e.g., birth and death, certificates of marriage, etc. (8) Hospital records: Routine entries, such as dates of admission and discharge, pulse, temperature, treatment given, etc., are admissible without oral evidence, but the cause of the disease or diagnosis are not accepted without oral testimony.

Chain of Custody of Evidence: It is a method to verify the actual possession of an object from the time it was first identified until it is offered into evidence in the Court room. Each specimen when obtained, should be labelled with the victim's name, the time and date, the nature of the specimen, identification number, and signed by the doctor. This information must be documented each time the material is handled by another person, and that person must give receipt for the material and will be included in the chain of custody. The evidence must not be damaged, contaminated, or altered in any significant way. The shorter the chain the better.

WITNESSES: A witness is a person who gives evidence regarding facts.

Types: (1) Common, and (2) Expert.

(1) Common witness is a person who gives evidence about the facts observed or perceived by him. He must show that he was capable of perceiving the fact by one of his own senses, and that he actually observed this fact. This principle is commonly known as the **"first-hand knowledge rule"**, which may be used to establish the exact circumstances of the case for the Court, e.g., A has seen B and C fighting with sticks on a certain road on particular day and time. In a case of traffic accident, the person who witnessed the accident becomes a common witness.

(2) EXPERT WITNESS is a person who has been trained or is skilled or has knowledge, experience or education in technical or scientific subject, and capable of drawing opinions and conclusions from the facts observed by himself, or noticed by others, e.g., **doctor, firearms expert, fingerprints expert, handwriting expert, etc. (S.45, I.E.A.).**

An expert witness may give his opinion (1) upon facts which are either admitted, or proved by himself or other witnesses at the trial, (2) on matters of common knowledge, and (3) on hypothetical questions (questions based on stated assumptions), based thereon. The answer should be given only after completely understanding the question. **The main obligation of an expert is to point out professional facts.** But, he may be asked to give professional estimate, which his observations seem to justify, e.g., whether or not an injury is caused in the manner alleged or from the causes assumed; the possible consequences; the reasonable estimate of damages, etc. **The opinion on a key question must be given in a guarded manner using terms, such as that the findings are "consistent with" an alleged form of trauma**, such as a fall against a hard object, or with the striking of the head with a blunt instrument of a particular type. It does not exclude other mechanisms which could reasonably or possibly have caused the same findings. **The doctor cannot change his opinion while giving evidence in a court, but he can indicate other possibilities**. Conclusions must be based on facts. **Conclusions are much more important than opinions**. The expert opinions based upon objective, indisputable facts, help to evaluate the reliability and credibility of other witnesses. **The expert evidence is of little value when there is a conflict of opinion between experts**. In such cases, the Courts usually accept that opinion which is not in conflict with direct evidence. **An expert witness may refer to books** to refresh his memory, or to correct or confirm his opinion **(S.159, I.E.A.)**. Books as such are not evidence, but if **an expert refers to specific passages as representing his views, they may be taken down as his own evidence**. A witness may adopt the views of any authority as his own, provided he has an honest belief in them. **The opinion of an expert can be proved false by standard books on the subject**.

A doctor can be both a common and expert witness. When he describes the wounds on the body, he acts as a common witness. But when he says that the wounds were antemortem or postmortem, or they were suicidal, homicidal or accidental, or gives opinion regarding the cause of death, he acts as an expert witness.

HOSTILE WITNESS is person who is supposed to have some interest or motive for concealing part of the truth, or for giving completely false evidence. The Court will declare a witness as hostile (adverse) on the suggestion of the lawyer of the party who has summoned the witness or prosecution lawyer. **On declaration of a witness as hostile, he can be cross-examined by the same-side lawyer.** Any of the above two types of witnesses can be hostile.

PERJURY: Perjury means wilful giving of false evidence by a witness while under oath, or failure to tell what he knows or believes to be true. It occurs if the person's earlier statement regarding the facts on oath and subsequent statement also must be on oath and both statements are opposed to each other and they cannot be reconciled. The witness is liable to be prosecuted for perjury, and the imprisonment may extend to 7 years.

RECORD OF EVIDENCE: The evidence of the witness is recorded in the following order **(S.138 to 159, I.E.A.)**.

(1) Oath: The witness has to take an oath in the witness box before he gives his evidence. He should take the oath as follows: "I do swear in the name of God, that what I shall state, shall be the truth, the whole truth, and nothing but the truth". If the witness is an atheist, he has to solemnly affirm instead of swearing in the name of God. **Oath is a declaration**

required by the law, which is compulsory and holds the witness responsible for consequences of his evidence. A child below 12 years is not required to take an oath.

(2) Examination-in-Chief (direct examination): This is the first examination of a witness. **It consists of questions put to him by the lawyer (counsel or advocate) for the side which has summoned him.** In criminal acts committed by an individual or group, the State becomes a party instead of the aggrieved person, and starts criminal prosecution, which is titled as "State versus A". **In a criminal trial, the burden to prove is always on the prosecution, and the accused is presumed to be innocent till the contrary is proved against him. In Government prosecution cases, the public prosecutor first examines the witness.** If witness is called by private party, he is first examined by the lawyer of that party. The **object is to elicit all relevant medical facts and the conclusions which the doctor has drawn from these facts.** Before giving evidence, it is advisable that the doctor meets the public prosecutor and discusses the previously prepared report, the certificate of death, photographs, etc., that the witness intends to show in the Court, and an outline or pattern should be worked out for the best way to elicit his testimony. The doctor should help the prosecutor in framing proper questions in proper sequence, so that all essential facts are elicited. If the witness intends to modify any of his conclusions, these should be pointed out to the lawyer. The lawyer will be able to advise the witness about the anticipated questions in cross-examination.

Leading questions are not allowed except in those cases, where the Judge is satisfied that a witness is hostile.

Leading questions: A leading question is one which suggests to the witness the answer desired, or which includes the answer implied in the question itself and admits of a conclusive answer by "Yes" or "No". "Was the length of cut 3 cm?". "Was it on the front of the abdomen?". "Was the injury caused by a sharp weapon?". They are all leading questions, as they suggest the answer "Yes" or "No". The proper questions should be: What was the length of the cut? Where was it seen? "What type of weapon would cause this injury?".

In examination-in-chief, the method of examination is by question and answer. The questions are usually short and demand some specific fact and short answer. But the answer can be given in narrative form, if it would be more informative and convincing. **The effect of the questions and answers is recorded indirectly, and not in the form of the question and answer.**

(3) Cross-examination: In this, **the witness is questioned by the lawyer for the opposite party, i.e., lawyer for the accused (defence lawyer). In a murder trial, the defence witness is cross-examined by public prosecutor.**

The main objects are: (1) to elicit facts favourable to his case, (2) to test the accuracy of the statements made by the witness, (3) to modify, or explain what has been said, (4) to develop new or old facts, (5) to remove any undue or excessive emphasis which may have been given to any of them, and (6) to discredit the witness. In doing this, the lawyer may try to weaken the evidence by showing that the evidence given is inaccurate, inconsistent, ill-founded, contradictory, and untrustworthy.

The cross-examination need not be limited to the facts to which the witness testified in his examination-in-chief. The competence and credibility of the witness is tested by questioning his qualifications, experience and the number of cases of the kind under consideration he has personally observed, and by testing his memory or power of observation. When material is available, the lawyer may attack the character of the witness. The witness may be asked questions on his bias or impartiality, his previous conviction, his reputation for untruthfulness, and on any handicap he may have, which would affect the reliability of his evidence. The Court will decide whether the witness should answer any particular question, when asked for a ruling. **The Judge can always disallow irrelevant questions.** The Court also has the power to disallow questions which are intended to insult or harass or offensive in form; but if they are relevant to the facts in issue, the Court cannot disallow them. **The witness has to answer any question relevant to the matter in issue, even though the answer will expose or prove his guilt directly or indirectly,** e.g., which would reveal that he has committed an illegal operation. If the question

is not understood, the witness should ask the lawyer to explain it better. **Answers favourable to the defence side should be given as promptly as those given on examination-in-chief. Leading questions are permissible during cross-examination. Cross-examination has no time limit,** and may last for hours or even days. Sometimes, cross-examination may act as a double-edged sword, i.e., it may damage the defence as much as the prosecution, especially if lawyer is not familiar with the subject, and the witness is efficient and honest.

(4) Re-examination (Re-direct examination): This is conducted by the lawyer for the side which has called the witness. **The object is to correct any mistake or to clarify or to add details to the statements the witness has made in cross-examination.** It is an opportunity for the witness to explain more fully some answer which might appear damaging to his direct evidence, because of skilful questioning or tactics by the cross-examiner. The witness should not bring in any new subject at this stage. The opposing lawyer has right of cross-examination on the new point raised. Leading questions are not allowed.

(5) Questions by Judge: The Judge may ask any question at any stage of the examination to clear up doubts. The Court is also empowered to recall and re-examine any witness already examined, if his evidence appears to the Court to be essential to the just decision of the Court.

The deposition of the witness is handed over to him. The witness after carefully going through it, is required to sign at the bottom of each page, and on the last page immediately below the last paragraph, and to initial any corrections. The witness should not leave the Court without taking permission of the Judge.

CONDUCT AND DUTIES OF THE DOCTOR IN THE WITNESS BOX

The following rules help a doctor in the witness box. (1) Be well prepared with the details of your evidence. (2) Take all records, relevant reports that may have to be quoted in the box, e.g., original notes, autopsy report, photographs, X-rays, toxicology and blood grouping reports. (3) Be well-dressed and modest. (4) Do not discuss the case with anyone in the Court, except the lawyer by whom you were asked to testify. (5) Stand up straight. (6) Be relaxed and calm and not frightened or nervous. (7) Be pleasant, polite and courteous to the lawyer. (8) Do not underestimate the medical knowledge of the lawyers. (9) Never attempt to memorise. The law allows to refresh your memory from copies of reports already submitted. (10) Listen carefully to the questions. Answer should be brief and precise and in the form of "Yes" or "No". The witness is not bound to answer yes or no, but may qualify his answer if it is necessary for accuracy and completeness. Answer only what is asked. (11) Do not fumble in referring to case notes, records, etc. (12) Avoid difference between your record and your testimony. (13) Speak slowly, distinctly and audibly to enable the Judge to record your evidence. Look people in the eye when you speak, it gives the impression of honesty. (14) Speak with assurance. Be confident but not overconfident or arrogant. (15) Use simple language, avoiding technical terms to the best of your ability. (16) Avoid superlatives and exaggerations, e.g., very large bruise, frightful injury, savage blow, most agonising pain, etc. (17) Address the Judge by his proper title, such as "sir" or "your honour". (18) Do not avoid a question. Say I do not know, if it is so, for no one can be expected to know everything. It indicates honesty. (19) Retain independence of your mind. Be honest, impartial, unbiased and truthful. Be frank to admit any points in favour of accused if that is the truth. (20) Never become hostile, angry, rude, or sarcastic during questioning. (21) Do not lose your temper. An angry witness is often a poor witness. Do not argue, just disagree if you do not agree; disagree firmly and repeatedly. (22) Do not over-emphasise replies to questions from the cross-examining lawyer. (23) Watch for double question and questions that include an assumption of facts which have not been proved. The answer to each part of the double question may be different. This should be made clear by the witness. (24) Express an opinion from your own knowledge and experience. Be prepared to give reasons for your opinion if asked. (25) Statements contained in older editions of text books need not be accepted as the medicine advances. Avoid speaking on a subject in which you have little or no practical experience. (26) A medical witness has no professional privilege, and therefore he must answer any question.

(27) Volunteering of information: Information should not be volunteered beyond that asked for in the question. **The answer should also not extend beyond the expert's knowledge.** Volunteered information is often not well prepared and is liable to cross-examination. As an expert witness, a medical practitioner may volunteer a statement, if he thinks

that injustice will result if he fails to make the statement.

MEDICOLEGAL MASQUERADES: Many cases of homicide go undetected because of the lack of suspicion and improper or inadequate investigation. **All cases of death should be regarded as unnatural, until proved otherwise.** Violent deaths may show minimal or no external evidence of injury, and conversely natural deaths can occur under such circumstances as to suggest falsely that violence was used. Accidental deaths and suicides can occur under circumstances which suggest homicide. In a suicide case, alterations may be made at the scene because of stigma. In a homicide case, the scene may be altered or rigged to suggest that death resulted from suicide or accident. The doctor must look for any possible inconsistencies between the apparent death scene and his actual scientific findings. In a case of hanging, the manner in which a ligature is applied to the neck, or the mode of suspension of a body may be determining features in the circumstances. In such cases, the real cause of death can be established by complete autopsy and police investigation.

INVESTIGATION OF THE SCENE OF DEATH

BASIC RULES: The basic rules for investigation of any scene of crime are: (1) verify that a crime has been committed, (2) look for signs of how it was committed, and (3) recover and preserve evidence that might lead to the arrest and conviction of the guilty.

THE DEAD DO TELL TALES.

The answers to the following questions have to be found: (1) Who is the victim? (identification). (2) When the death and injuries occurred? (time of death and injuries). (3) Where the death occurred? (scene and circumstances of death). (4) What injuries are present? (description of injuries). (5) Which injuries are significant? (major, minor, true, artefacts, postmortem injuries). (6) Why and how injuries were produced? (mechanism and manner of death, i.e., natural, accidental, suicidal or homicidal). If unnatural, determine the means or agent causing death, e.g., knife, firearm, poison, etc. and if homicide assist in identifying the person responsible for death.

CONDUCT AND DUTIES OF THE DOCTOR AT THE SCENE OF CRIME: Complete and accurate recording of the scene as it was found is very important. This can be done by accurate diagrams, notes and photography. The scene may show evidence of a struggle, and on the body vital trace evidence may be present. The examination at the scene should be limited to a search for such evidence which might be dislodged or possibly lost during the transfer of the body to the mortuary.

(1) If the victim of an assault is living when first seen, the doctor must do everything to save the life. (2) If death is imminent, he should obtain a dying declaration. (3) Retain any material which is relevant, e.g., in cases of suspected poisoning, he must retain vomit, left-over poison, drinking utensils, etc. (4) He must make sure that death has occurred. (5) If he suspects foul play, the police should be informed. (6) He must obtain all possible information regarding the crime. (7) He must identify the body, which should also be identified by the relatives and the police. (8) He must enquire whether the body has been moved at all before he first saw it. (9) He should ask the investigating officer before moving anything. Photograph the scene from several angles. He should follow but not lead the police around the scene. (10) He should not give opinion without proper thought. (11) He should make adequate notes: (a) Date, time, address or location. (b) Name and sex of deceased. (c) A list of all persons present. (d) General observation about the scene; any evidence of struggle, such as overturned furniture or trampled ground. Note surroundings of the body, such as walls, flooring, fixtures, furniture, doors and windows. If outside, record the type of weather, the state of ground and the vegetation present. (e) Temperature of the surroundings, and the rectal temperature of the deceased should be taken. (f) Make a sketch noting such points of importance as direction and position of blood (pools or splashes), position of the body and any weapons. The distribution of blood stains and their shape which may point to the site of injury should be noted. Note the amount of bleeding at the scene. Describe the clothing and note any tears, cuts, missing buttons, etc. If the weapon is in the hand of the deceased, note whether it is loosely held or tightly grasped. Examine the hands and forearms for defence wounds. Make note of injuries and record them on body diagrams. (g) Position and appearance of the body, rigor mortis, postmortem lividity, etc., which assist in estimating the time of death. (h) Free hair, fibers or other foreign matter which is likely to be dislodged when the body is moved, should be searched and removed with adhesive tape. (i) If there are any bite marks, they should be swabbed with a cotton wool swab moistened with saline. (j) The pubic hair should be combed in situ in cases of sexual assault, and loose hair collected. (k) Examine the objects on premises, e.g., dates on mail and newspapers, condition of food on table, etc., to determine the time of death. (l) Photograph any ligature before removal, cut if necessary leaving the knot or knots intact. (m) If a weapon is found, handle it with

care to preserve fingerprints, blood stains, hair, fibres, etc. (n) Leave firearms in the condition they are found. (o) Note position of each bullet and casing. Bullets, etc., should be marked for identification. (12) Do not touch anything, especially a possible weapon. (13) Do not smoke or throw cigarette stubs, matches, etc., as trace evidence including small items discarded by a suspect, may be important in the proof of the crime. (14) The scene should be examined by fingerprint examiner, followed by a trace evidence specialist. (15) Transfer the body to the mortuary wrapped in a large sheet of plastic or clean cloth.

DISADVANTAGES of the doctor not visiting the scene are: (1) When the body is transferred to mortuary, fresh abrasions may be produced on it during transit. (2) Clothing will be disarranged, blood stains will form on parts of clothes originally free from them. (3) Fresh tears in clothes may be produced by rough handling. (4) Existing rigor mortis may be broken down at least partially.

BASIC RULES FOR PRESERVATION OF MEDICOLEGAL EVIDENCE: For evidence to be legally accepted by the Courts: (1) It must be obtained in a legal manner. (2) It must be relevant to the issue. (3) The chain of custody of the item must be intact and known. (4) It must be evaluated by the qualified experts.

COLLECTION OF EVIDENCE: COLLECT: (1) Every article even remotely likely to be helpful in the investigation. (2) Any item likely to carry fingerprints. (3) Use separate container for each item. (4) Every article collected must bear identifying marks either (a) direct, in which marks are put on the item of evidence itself, or (b) indirect, in which notations of identification are placed on a container in which the evidence is placed.

PROCEDURE OF CRIMINAL TRIAL

The proceedings of investigations by the police in criminal offences are sent to the Judicial Magistrate of the area. **All offences punishable with death, imprisonment for life, or for a term exceeding 2 years are tried as warrant cases. All other cases are tried as summons cases (non-cognisable offences).**

SESSIONS TRIAL: In cases submitted by the police, when the accused is brought before the Court, the Magistrate must first make sure that the accused has received all the required documents. The Magistrate considers the documents, examines the accused, and hears the prosecution and defence. If he finds the charge groundless, he records his reasons and discharges the accused. **If the Magistrate is of the opinion that the accused should be committed to trial by Sessions Court, he frames a charge of the offence, which is read and explained to the accused, and makes an order committing the accused to trial by the Court of Sessions,** and records briefly the reasons for such commitment. A warrant is issued to keep the accused in custody. The Magistrate sends the charge, the record of enquiry and any weapon or other thing which is to be produced in evidence, to the Court of Sessions. At the first hearing, the public prosecutor opens the case by describing the charge against the accused and stating the evidence by which he proposes to prove the accused guilty of the offence charged. After going through the record, and hearing the submissions of the defence and the prosecution, if the Judge considers that there is no sufficient ground for proceeding against the accused, he discharges him recording his reasons for doing so. Otherwise, the Judge frames a charge which is read out and explained to the accused, and his plea is recorded. If he pleads guilty, he may be convicted, otherwise a date is fixed for hearing. The prosecution witnesses are called. Each witness is examined by the prosecutor, and may be cross-examined by the defence, and re-examined by the prosecution, as well as questioned by the Court. If, after hearing the prosecution evidence, examining the accused, hearing the prosecution and the defence, the Judge considers that there is no evidence that the accused committed the offence, he passes an order of acquittal. If the accused is not acquitted, he is asked to defend himself. Any written statement submitted by the accused is filed with the record. The defence witnesses are then called. Each witness is examined by the defence lawyer, and may be cross-examined by the prosecutor and re-examined by the defence, and may be questioned by the Judge. The evidence of all the witnesses is recorded in writing either by the Judge himself or by his dictation in open Court. At the conclusion of the evidence, the prosecution sums up the case and the accused or his lawyer is entitled to reply and the prosecutor may enter his submissions if permitted by the Judge. If the accused is found guilty he is convicted, otherwise he is acquitted. Both the prosecution and defence can appeal to the High Court against an acquittal or conviction. When the Court of Sessions passes the sentence of death, the proceedings are submitted to the High Court for confirmation of the sentence.

CHAPTER

3 Medical Law and Ethics

The medical profession is governed by legislation and by a Code of Ethics and Etiquette. The broad principles of medical ethics are formulated by National and State Medical Councils and the World Medical Association.

HOMOEOPATHY CENTRAL COUNCIL ACT, 1973: The Act was passed to regulate the conduct of homoeopathic practitioners in India and to constitute central council. The Act empowers the Central Government to constitute the Central Homoeopathy Council. The council is empowered to prescribe uniform standards for admission, curriculum, syllabus and duration of course for professional training in Homoeopathy. It also provides for the maintenance of a Central Register of practitioners and a Code of Ethics for them. It recognises foreign homoeopathic qualifications in its third schedule.

HAHNEMANNIAN OATH: On my honour, I swear that I shall practice the teachings of Homoeopathy, perform my duty, render justice to my patients and help the sick whosoever comes to me for treatment. May the teachings of my master hahenemann, inspire me and may I have the strength for fulfilment of my mission.

INDIAN MEDICINE CENTRAL COUNCIL ACT, 1970: It was enacted to lay down minimum standards of education and practice of Indian system of medicine. These include Ayurveda, Siddha, Unani Tibbi and Ashtanga. As per the provisions of this Act, the Central Council for Indian Systems of Medicine has been authorized to prescribe qualifications for practitioners of these systems of medicine. As per this Act, no person shall be eligible to practice Indian Medicine in any state or authenticate legal certificates without the recognized qualifications [Sec. 17 (b), Indian Medicine Central Council Act, 1970]. The Act contains three schedules, which give a detailed description of the allocation of seats in the Central Council to the various systems of Indian Medicine.

INDIAN MEDICAL DEGREES ACT, 1916: This Act was passed to regulate the grant of titles implying qualification in Allopathic system of Western Medical Science. Certain authorities mentioned in the Act have the right to grant medical degrees, diplomas, licenses and certificates, implying that the grantee or recipient thereof is qualified to practise Western Medical Science. The false assumption and use of a medical title are punishable under the Act.

MEDICAL COUNCIL OF INDIA ACT, 1956: The Act of 1933, was repealed and the Act of 1956, was passed.

Constitution: The Medical Council consists of a number of members of the profession elected from each State, each university and some are nominated by the Central Government. They hold office for a term of five years. A President and a Vice-President are elected from among these members. The Council appoints a Registrar, who acts as Secretary and looks after the routine work. The Executive Committee consists of president, Vice-President and seven to ten other members. Other committees for general or special purposes are formed from among its members.

Schedules: The First Schedule of the Act contains the recognised medical qualifications granted by universities in India; the Second Schedule those granted outside India. Part I of the Third Schedule contains those granted by medical institutions in India, but not included in the First Schedule; part II of the Third Schedule those granted outside India, but not included in the Second Schedule.

MEDICAL COUNCIL OF INDIA

Functions: (1) Medical Register: The Council maintains a register of medical practitioners, known as the Indian Medical Register. **It contains the names of all persons who are enrolled on any State Medical Register.** If the name of a person enrolled on a State Medical Register is removed from the Register, the Council also removes such person's name from the Indian Medical Register.

(2) Medical Education: The Council prescribes standards of postgraduate medical education for the guidance of the universities. It may advise the universities in maintaining uniform standards for

postgraduate medical education throughout India. A Postgraduate Medical Education Committee, consisting of nine members is formed for this purpose. **Prior approval of Medical Council of India is necessary before starting a new medical college or to increase the number of seats, and a new postgraduate medical course in any discipline.**

The Council maintains the standards of undergraduate medical education. It prescribes the minimum standards of medical education required for granting recognised medical qualifications by universities or medical institutions in India. It appoints Medical Inspectors to attend at any or all examinations held by universities or institutions in India for the purpose of recommending to the Central Government recognition of medical qualifications. The Inspectors have no power to interfere with the conduct of any training or examination. **The Inspectors report to the Council on the adequacy of the standards of medical education including staff, equipment, accommodation, training, and other facilities prescribed for giving medical education** and on the sufficiency of every examination they attended. The Committee forwards a copy of any such report to the university or medical institution concerned for its remarks. A copy with the remarks of the university or institution is sent to the Central Government. **If the Council is not satisfied with the standards, it can make a representation to the Central Government to withdraw recognition of any medical qualifications of any college or university.** The council on getting assurance for future rectifications of all deficiencies by the institution can send its recommendation to the Central Government for reconsideration. The Central Government will again refer it to the council, which in turn will send its inspectors to visit the institution for verifying the implementation of assurances, and will send the report accordingly. Any university which grants a medical qualification not included in the First Schedule, may apply to the Central Government to have such qualification recognised. The Central Government, after consulting the Council may by notification in the Official Gazette, amend the First Schedule so as to include such qualification therein.

Medical qualifications granted by unrecognised institutions are not held at par with qualifications recognised by MCI, as regards further training and other facilities.

(3) Recognition of Foreign Medical Qualifications: The Council provides for the registration of any citizen of India, who has obtained foreign medical qualifications which are not recognised under the existing Act. **It has the power to negotiate with the authority in any State or country outside India for the reciprocal recognition of medical qualifications.** The Central Government may amend the Second Schedule to include any medical qualification, which the Council has decided should be recognised.

(4) Appeal against Disciplinary Action: If the name of any person is removed from the State Medical Register, he may appeal to the Central Government, after exhausting all the remedies under the State Act. **The decision of the Central Government, which is given after consulting the Indian Medical Council, is binding on the State Government and the State Medical Council.**

(5) Warning Notice: The Council may prescribe standards of professional conduct and etiquette, and a Code of Ethics for medical practitioners. It can issue Warning Notice containing certain practices which are regarded as falling within the meaning of the term "serious professional misconduct".

(6) It is empowered to issue certificates of good conduct and character to medical students/doctors going abroad for higher studies/service.

STATE MEDICAL COUNCILS

Constitution: The State Medical Councils are autonomous bodies, established under the State Medical Council Act. Each of these Medical Councils consists of members elected by the registered medical practitioners and those nominated by the State Government. The President and the Vice-President of the Council are elected by the members from among themselves.

Functions: (1) Medical Register: The Council appoints a Registrar, who keeps a Register of medical practitioners. **Any person having any of the recognised medical qualifications can get his name registered.** The name, residence, qualifications, and the date on which each qualification was granted are entered in the Register, on payment of prescribed fees. After passing the qualifying examination, it is necessary to undergo a period of training, before such qualification is granted to him. A provisional registration in a State Medical Register is granted to

such person on application, to enable him to practice medicine in an approved institution for the required period. **Any additional qualification obtained later can also be registered.** The Registrar should inform the Indian Medical Council without delay, of all additions and other amendments in the State Medical Register, made from time to time.

Declaration of Geneva: The modernised version of Hippocratic Oath is the Declaration of Geneva (1948), as adopted by the World Medical Association. **At the time of registration by the State Medical Council, each applicant is given a copy of the Declaration of Geneva (amended in 2006) by the Registrar concerned.**

I shall abide by the code of medical ethics as enumerated in the Indian Medical Council (Professional conduct and etiquette and ethics) regulations, 2009: (1) I solemnly pledge to consecrate my life to the service of humanity. (2) I will give to my teachers, the respect and gratitude that is their due. (3) I will practise my profession with conscience and dignity. (4) The health of my patient will be my first consideration. (5) I will respect the secrets that are confined to me, even after the patient has died. (6) I will maintain by all the means in my power, the honour and the noble tradition of the medical profession. (7) My colleagues will be my sisters and brothers. (8) I will not permit considerations of age, disease or disability, creed, ethnic origin, gender, nationality, political affiliation, race, sexual orientation, social standing or any other factor to intervene between my duty and my patient. (9) I will maintain the utmost respect for human life. (10) I will not use my knowledge to violate human rights and civil liberties even under threat.

The medical man is to affirm that I make these promises, solemnly, freely and upon my honour.

(2) Disciplinary Control: They have the power to remove the names of medical practitioners permanently or for a specific period from their Registers, when after due enquiry they are found to have been guilty of serious professional misconduct. It has also authority to direct the restoration of any name so removed.

(3) It can issue **warning notice** similar to those of the Indian Medical Council.

Judicial Procedure of State Council: Complaint: The proceedings are started (1) when information reaches the office of the Council that a registered medical practitioner has been convicted of a cognisable offence or has been censured by judicial or other competent authority in relation to his professional character, or has been found guilty of conduct which *prima facie* constitutes serious professional misconduct. (2) by a complaint being made by some person or body against the practitioner. (3) Suo moto the Council has the same powers as Civil Courts under Code of Civil Procedure, 1908. This makes all enquiries on the misconduct of doctors to be judicial proceedings.

Council Procedure: The complaint is referred to the subcommittee or to the Executive Committee which considers the complaint, causes further investigation and takes legal advice. If no *prima facie* case is made out, the complainant is informed accordingly. **If an enquiry is to be made, a notice is issued to the practitioner specifying the nature and particulars of the charge, and directing him to answer the charge in writing, and to attend before the Council on the appointed day.** At the hearing, the complainant or his legal advisor and the practitioner must be present. After the conclusion of evidence, vote is taken, and the judgement given. **If the majority vote confirms that the charge has been proved, the Council must then vote again and decide whether the name of the practitioner should be removed from the Register, or whether he should be warned not to repeat the offence.**

Erasure of Name: The name of the doctor is removed from the Medical Register: (1) After the death of the registered practitioner. (2) Entries which are made in error or as a result of fraud. (3) **Penal erasure:** The main cause for erasure is serious professional misconduct, and this is known as penal erasure. It is sometimes termed **"the professional death sentence". It deprives the practitioner of all the privileges of a registered practitioner.**

Serious Professional Misconduct (Infamous conduct in professional respect)**: It is any conduct of the doctor which might reasonably be regarded as disgraceful or dishonourable.** The conduct of the doctor is judged by professional men of good repute and competence.

WARNING NOTICE

A registered medical practitioner is required to observe certain prescribed rules of conduct contained in Code of Medical Ethics, published by the Medical

Council of India and by many State Medical Councils. **The Council gives examples of offences which constitute professional misconduct, but it stresses the fact that it is not a complete list.** The Council can also consider any form of alleged professional misconduct, which does not come within any of the examples contained in the Warning Notice. Each case has to be decided on its specific facts and merits. **If anyone is found guilty of any of the following offences mentioned in the Warning Notice issued by the Medical Council of India, his name can be erased from the Medical Register.**

(1) **Adultery** (voluntary sexual intercourse between a married person, and a person married or not, other than his or her spouse). (2) Improper conduct or association with a patient or member of the patient's family. (3) Conviction by a Court of Law for offences involving moral turpitude. (4) Issuing a false, misleading or improper certificate in connection with sick benefit, insurance, passport, attendance in Court, public services, etc. (5) Withholding from health authorities information of notifiable diseases. (6) Performing or enabling an unqualified person to perform an abortion or any illegal operation for which there is no indication. (7) Violating the provisions of the Drugs Act. (8) Selling Scheduled Poisons to the public under cover of his qualifications, except to his patients. (9) **Dichotomy or fee-splitting, i.e., receiving or giving commission or other benefits to a professional colleague or manufacturer or trader in drugs or appliances or a chemist, dentist, etc.** (10) Using of touts or agents for procuring patients. (11) Disclosing the secrets of a patient that have been learnt in the course of his professional work, except in a Court of Law. (12) **Covering, i.e., assisting someone who has no medical qualification to attend, treat or perform an operation on some person in respect of matters requiring professional discretion or skill.** (13) Association with manufacturing firms: (a) A physician should not have any personal ownership in patents for any drug, apparatus, or instrument used in medicine or surgery. He should not ask or receive rebates or commission from prescribing of any agent used therapeutically. (b) A physician must not write prescriptions in private formulae of which only he or a particular pharmacy has the key. He can keep certain lotions or mixtures as long as the formulae of the same are available. (14) **Advertising:** (a) A physician should not publish on his own in the lay press, report of cases treated or operated on by him. He should not sanction the publication of any certificates for drugs, foods, or appliances issued by him. (b) A physician can write to the lay press under his own name on matters of public health, hygienic living, or to deliver public lectures, give talks on the radio for the same purpose. (c) A physician can announce in lay or professional press, his starting of practice, interruption or restarting of it after a long interval, or a change of his address, but such an announcement shall not appear more than twice. (d) An institution, such as a maternity home, a sanatorium, a house for the crippled or blind, etc., may be advertised in the lay press but the name of the doctor should not appear. (e) It is improper for a physician to use an unusually large signboard and to write on it anything other than his name, qualification and name of his speciality. The same should be the contents of his prescription paper, which may in addition contain his registration number, address and telephone number. It is improper to keep a signboard on a chemist's shop or in places where he does not reside or work. (f) A physician must not exhibit publicly the scale of fees, except in his consulting or waiting room. (g) He should not advertise himself through manufacturing firms directly or indirectly. (15) Professional association with bodies or societies of unqualified persons formed for the purpose of turning unqualified practitioners. (16) Running an open shop for sale of medicines, for dispensing prescriptions of other doctors or for sale of medical or surgical appliances. Manufacturing or sale of proprietary medicines, whose formulae are not displayed in the label. (17) A physician may patent surgical instruments, appliances and medicines or copyright publications, methods and procedures. The use of such patents or copyrights or the receipts of remuneration from them, which retards or inhibits research or restrict the benefit derivable therefrom are unethical. (18) Refusal to give professional service on religious grounds. (19) Drunk and disorderly so as to interfere with proper skilled practice of medicine. (20) A physician shall not aid or abet torture. (21) Sex determination test shall not be done with intent to terminate the life of a female foetus. (22) Failure to obtain consent of both husband and wife in an operation which may result in sterility. (23) No

act of *in vitro* fertilization or artificial insemination shall be undertaken without informed consent of the woman, her husband and donor. (24) A physician should observe the laws of the country in regulating practice of medicine, and will not assist others to evade such laws. **The important offences may be described as the "6 As". (1) Adultery, arising out of professional relationships. (2) Advertising. (3) Abortion (unlawful). (4) Association with unqualified persons in professional matters. (5) Addiction. (6) Alcohol.**

Rights and Privileges of Registered Medical Practitioners: (1) Right to practise medicine. (2) Right to choose a patient. (3) Right to dispense medicines. (4) Right to possess and supply dangerous drugs to his patients. (5) Right to add title, descriptions, etc., to the name. (6) Right to recovery of fees. (7) Right for appointment to public and local hospitals. (8) Right to issue medical certificates. (9) Right to give evidence as an expert.

RED CROSS EMBLEM: The Geneva Convention Act (1960), prohibits use of Red Cross and other allied emblems such as "Red Crescent", "Red Crystal" for any purpose without approval of the Government of India, and imposes a penalty on anyone who uses such emblems. **It is the right, only of members of medical service of any army.**

DUTIES OF MEDICAL PRACTITIONERS

(I) Duty to Exercise a Reasonable Degree of Skill and Knowledge: The duty of care arises simply by examining someone for signs of illness or trauma or even by accepting a patient onto a list of existing patients. As soon as a doctor gives advice and counselling over the telephone, a legal duty to the patient arises. If no advice is given, no duty arises. He owes this duty to the child even when engaged by his father. He owes this duty even when patient is treated free of charge. It neither guarantees cure nor an assured improvement. A practitioner is not liable because some other doctor of greater skill and knowledge would have prescribed a better treatment, or operated better in the same circumstances.

(II) Duties with regard to Attendance and Examination: When a practitioner agrees to attend a patient, he is under an obligation to attend to the case as long as it requires attention. He can withdraw only after giving reasonable notice, or when he is asked by the patient to withdraw. He cannot withdraw without the consent of patient except for valid reasons, e.g., the patient is not following his instructions, using medicines not prescribed by him, malingering or using intoxicants or poisons. If a physician is unable to treat the patient when his services are needed, he may provide a qualified and competent substitute doctor. If the practitioner cannot cure a patient, he need not withdraw, if the patient desires his services. **He should not get his patient examined or operated upon by another doctor without his permission.** If the doctor is called by the police to attend a case of accident, he may render first aid and advice, but here no doctor–patient relationship is established. **Legally, a doctor need not accept as patients all who come to him for treatment, but ethically in an emergency, the doctor should not refuse to treat a patient.**

(III) Duty to Furnish Proper and Suitable Medicines: If the doctor has his own dispensary, he should furnish suitable medicines to the patient. Otherwise, **he should give a legible prescription, mentioning full and detailed instructions.** The doctor is held responsible for any temporary or permanent damage in health, caused to the patient due to wrong prescription.

(IV) Duty to give Instructions: The doctor should give full instructions to his patients or their attendants, regarding the use of medicines and diet. He should mention the exact quantities and timing for taking medicines. **Patients should be instructed regarding the adverse reactions** and to stop the drug in case of reaction, and to approach the doctor immediately.

(V) Duty to Control and Warn: A physician has to warn patients of the dangers involved in the use of a prescribed drug or device. If the doctor fails to inform the patient of the known or reasonably foreseeable dangerous effects of a drug or device, he becomes liable not only for the harm suffered by the patient, but also for injuries his patient may cause to third parties. Similarly, when a doctor detects a medical condition that may impair the patient's ability to control his activities, the doctor has a duty to warn the patient, family, employer or authorities.

(VI) Duty to Third Parties: If a patient suffers from an infectious disease, the doctor should warn

not only the patient, but also third parties known to be in close contact with patient, e.g., relatives, friends, co-workers.

(VII) Duty towards Children and Adults incapable of taking care of themselves: When applying hot water bottles and similar articles to children, special care should be taken, for the child may be injured. Special precautions should be taken in case of adults who are incapable of taking care of themselves due to insanity or some physical disability.

(VIII) Duty to Inform Patient of Risks: A mentally sound adult patient must be told of all the relevant facts. But under certain conditions arising out of psychological factors, some facts have to be withheld.

(IX) Duty with Regard to Poisons: Poisons should be handled carefully. When a doctor is called upon to treat a case of poisoning: (1) he should give immediate treatment, and (2) he should **assist the police in determining whether the poisoning is accidental, suicidal or homicidal.**

(X) Duty to Notify Certain Diseases: A doctor is bound to give information of communicable diseases (smallpox, chickenpox, cholera, plague, typhoid, measles, diphtheria, yellow fever, food poisoning), births, deaths, etc., **to the Public Health Authorities.**

(XI) Duties with Regard to Operations: He should explain the nature and extent of operation, take proper care to avoid mistakes, must keep abreast of current standard practice, operate with proper and sterilised instruments, must not experiment, make sure that all the swabs, instruments, etc., put in are removed, should take proper postoperative care, and should give proper directions to his patient when discharging him.

(XII) Duties under Geneva Conventions: The wounded or sick of the armed forces, ship-wrecked persons, prisoners of war, or civilians of enemy nationality should be treated without any adverse distinction based on sex, race, nationality, etc.

(XIII) Duties with Regard to Consultation: Consultation should be advised preferably with a specialist, if the patient requests consultation, in an emergency, when an operation involving danger to life or affecting vitality of intellectual or generative functions is to be undertaken, when the case is obscure or has taken a serious turn, in a criminal assault, homicidal poisoning, etc.

(XIV) Duty in connection with X-ray Examination: As far as possible, all cases of accident, unless they are very minor should be X-rayed. The radiologist should take precautionary measures against X-ray burns, pain or scars and other complications while giving therapy to patient.

(XV) PROFESSIONAL SECRECY (Confidentiality): It is an implied term of contract between the doctor and his patient. The doctor is obliged to keep secret all that he comes to know concerning the patient in the course of his professional work. Everything said by a patient or his family members to a physician in the context of medical diagnosis and treatment is confidential. **Its disclosure would be a breach of trust and confidence. The patient can sue the doctor for damages** (mental suffering, shame or humiliation), if the disclosure is voluntary, has resulted in harm to the patient, and is not in the interest of the public.

Examples: (1) A doctor should not discuss the illness of his patient with others without the consent of the patient. (2) If the patient is a major, the doctor should not disclose any facts about the illness without his consent to parents or relatives even though they may be paying the doctor's fees. In the case of a minor or an insane person, guardians or parents should be informed of the nature of the illness. (3) A doctor should not answer any enquiry by third parties, even when enquired by near relatives of the patient, either with regard to the nature of the illness or with regard to any subsequent effect of such illness on the patient, without the consent of the patient. (4) A doctor should not disclose any information about the illness of his patient, without the consent of the patient, even when requested by a public or statutory body, except in case of notifiable diseases. (5) Even in the case of husband and wife, the facts relating to the nature of illness of the one, must not be disclosed to the other, without the consent of the concerned person. (6) In divorce and nullity cases, no information should be given without getting the consent of the person concerned. (7) Medical Officers in Government service are also bound by the code of professional secrecy, even when the patient is treated free. (8) When a doctor examines a Government servant on behalf of the Government, he cannot disclose the nature of illness to the Government without the patient's consent. (9) When a domestic servant is examined at the request of the master, the

doctor should not disclose any facts about the illness to the master without the consent of the servant, even though the master is paying the fees. (10) The medical officer of a firm or factory should not disclose the result of his examination of an employee to the employer without the consent of the employee. (11) A person in police custody as an under-trial prisoner has the right not to permit the doctor who has examined him, to disclose the nature of his illness to any person. If a person is convicted, he has no such right and the doctor can disclose the result to the authorities. (12) In reporting a case in any medical journal, care should be taken that patient's identity is not revealed from the case notes or photographs. (13) In the examination of a dead body certain facts may be found, the disclosure of which may affect the reputation of the deceased or cause anguish to his relatives, and as such, the doctor should maintain secrecy. (14) The medical examination for taking out life insurance policy is a voluntary act by the examinee and therefore, consent to the disclosure of the finding may be taken as implied.

PRIVILEGED COMMUNICATION: It is a statement made bonafide upon any subject matter, by a doctor to the concerned authority, due to his duty to protect the interests of the community or of the State. To be privileged, the communication must be made to a person having interest in it, or in reference of which he has a duty. If made to more than one person, or to a person who has not a direct interest in it, the plea of privilege fails. The doctor should first persuade the patient to get his consent before notifying the proper authority. If the doctor discloses professional secrets for the purpose of protecting the interest of the community (under a moral and social obligation), **he will not be liable to damages. The following are the examples of privileged communication or in other words, exceptions to the general rule of professional secrecy.**

(1) Infectious Diseases: If a patient suffering from an infectious disease is employed as cook or waiter in a hotel, or a food-handler with an enteric infection, or a teacher with tuberculosis or other infective disease, or as children's nurse, etc., he should be persuaded to leave the job until he becomes noninfectious. If the patient refuses to accept this advice, the doctor can inform the employer.

(2) Venereal Diseases: If a person is suffering from syphilis and is about to marry, it is the duty of the doctor to advise the patient not to marry till he is cured; if the person refuses, the doctor can disclose the syphilitic condition of the patient to the woman concerned or to her parents. Swimming pools should be prohibited to those having syphilis or gonorrhoea; but if the person refuses, the authorities can be informed. The doctor can inform the warden of a hostel, if any boarder is suffering from venereal disease.

(3) Servants and Employees: An engine driver or a bus driver may be suffering from epilepsy, high blood pressure, alcoholism, drug addiction, or colour blindness. The doctor should persuade the patient to change his employment, because of the danger of his present occupation, both to himself and to the public. If this fails, the doctor should inform the employer, that the patient is unfit for that kind of employment.

(4) Notifiable Diseases: A medical practitioner has a statutory duty to notify births, deaths, infectious diseases, etc., to the Public Health authorities.

(5) Patient's own Interest: The doctor should disclose to the parents or guardians of signs in the patient of melancholia, suicidal tendencies, etc., so that proper care may be taken.

(6) Self-interest: Both in civil and criminal actions by the patient against the doctor, evidence about the patient's condition may be given.

(7) Courts of Law: **In a Court of law, a doctor cannot claim privilege concerning communications between himself and his patient, if it is relevant to the inquiry before the Court.** In all cases, the doctor should appeal to the Judge, before disclosing a professional secret. The doctor may request the Judge that he will give the information in writing, so that it is not made public. The witness should not voluntarily disclose information either in Court or out of it, but **for the actual evidence demanded by the Court, he is protected from civil action against breach of confidence.**

A doctor can disclose and discuss the medical facts of a case with other doctors and paramedical staff, such as nurses, radiologist, physiotherapist, etc., to provide better service to the patient.

(8) Suspected Crime: If the doctor learns of a serious crime, such as murder, assault, rape, etc., by treating the victim or assailant, he is bound to give

information to the police (S.39, Cr.P.C). Thus, if a doctor treats a person suffering from gunshot or stab wounds due to criminal assault, he must inform the police.

Physician's Responsibility in Criminal Matters: In medicolegal cases, treatment gets priority. Thereafter procedural criminal law will operate. A doctor who is aware of the commission of crimes, such as murder, dacoity, waging war against the lawful Government, helping the escape of prisoners, etc., is legally bound to report them to the nearest Magistrate or police officer (**S.39, Cr.P.C., S.176, I.P.C.**). The doctor knowing or having reason to believe that an offence has been committed by a patient whom he is treating, intentionally omits to inform the police, shall be punished with imprisonment upto 6 months (**S.202, I.P.C.**). The practitioner should hand over to the police a man whom, from the nature of injury, he may suspect to be an assailant in a murder case. But, if he treats a person who has attempted to commit suicide, he is not legally bound to report, but if the person dies he has to inform the police. The practitioner's responsibility in case of criminal abortion and poisoning has been described in the concerned chapters.

SPECIAL DUTY OF A DOCTOR IN EMERGENCY CASES: In emergency he has moral, ethical and humanitarian duty to do his best to help the patient in saving his life. In medicolegal injury cases a doctor is obliged to give necessary medical aid to save the life of the patient and render all help to see that the person reaches the proper expert/institution as early as possible.

DUTIES OF A PATIENT: (1) He should furnish the doctor with complete information about past illness, and family history of diseases and the facts and circumstances of his illness. (2) He should strictly follow the instructions and directions of the doctor as regards diet, medicine, mode of life, etc. (3) He should pay a reasonable fee to the doctor.

PROFESSIONAL NEGLIGENCE (MALPRACTICE)

Professional negligence is defined as absence of reasonable care and skill, or wilful negligence of a medical practitioner, in the treatment of a patient which causes his bodily injury or death. Negligence is defined as doing something that one is not supposed to do, or failing to do something that one is supposed to do.

(1) CIVIL NEGLIGENCE: The question of civil negligence arises: (1) When a patient, or in case of death, any relative brings suit in a civil Court for getting compensation from his doctor, if he has suffered injury due to negligence. (2) When a doctor brings a civil suit for obtaining of his fees from the patient or his relatives, who refuse to pay the same alleging professional negligence.

Elements of negligence: Liability for negligence arises if the following conditions are satisfied: **(1) Duty:** Existence of a duty of care by the doctor. **(2) Dereliction: The failure on the part of the doctor to maintain applicable standard of care and skill. (3) Direct causation:** The failure to exercise a duty of care must lead to damage. The patient must show that a reasonably close and causal connection exists between the negligent act or omission and the resulting injury without any intervening cause. This is referred to as **legal cause or proximate cause. (4) Damage: The damage should be of a type that would have been foreseen by a reasonable physician.**

Burden of proof: The patient should prove all four elements of negligence by a preponderance of the evidence. It requires enough proof to show that each of the four elements of a negligent claim is true.

Damage (injury): Absence of proper skill or care that prolongs his illness, causes physical harm, bodily impairment, disfigurement or increases his suffering, diminishes his chances of recovery, causes the patient's death, constitutes injury in a legal sense. **The patient cannot sue a doctor for negligence, if no damage has occurred. The patient must suffer some loss, which can be measured and compensated in terms of money.**

Liability: The amount of damage done is a measure of the extent of the liability. Some **examples are**: (1) Medical expenses including medical rehabilitation, vocational rehabilitation, retraining or other incidental expenses like transportation, additional surgical procedures, daily nursing care and medications for a severely brain-damaged baby for the remainder of baby's life. (2) Loss of earning, either due to enforced absence from work, or prevention or reduction of his ability to pursue his occupation. (3) Pain and suffering, either physical or mental. (4) Reduced enjoyment of life, such as loss of limb or sense. (5) Loss of potency. (6) Reduction in expectation of life. (7) Aggravation of a pre-existing condition. (8) Death.

Instances of Medical Negligence: (1) Refusal to admit patients requiring urgent hospitalisation. (2) Failure to obtain informed consent. (3) Failure to examine patient himself. (4) Failure to perform sensitivity tests and immunise when indicated. (5) Not ordering X-ray examination where the history suggests the possibility of a fracture, or dislocation or presence of a foreign body in a wound. (6) Not reading the X-ray film correctly or in failing to get it read by a competent person. (7) Failure to attend the patient in time, or failure to attend altogether. (8) Failure or delay in diagnosis. (9) Making a wrong diagnosis due to absence of skill or care. (10) Failure to provide a substitute during his absence. (11) To delegate his duty of treating or operating upon a patient to another doctor without the consent of the patient. (12) Failure to keep well informed of advances in medical sciences. (13) Failure to warn the patient of side-effects. (14) Failure to obtain consultation where appropriate. (15) Experimenting on patient without consent. (16) Giving overdose of and giving poisonous medicines carelessly. (17) If his negligence causes others to catch a disease from his patient. (18) Negligent management of procedures. (19) Continue a practice regarding which several warnings as to its dangers have been given. (20) Failure to give proper instructions. (21) Failure to give proper postoperative care. (22) Iatrogenic medical complications during diagnosis or treatment. (23) Prematurely discharging the patient. (24) To cover up an error of judgement.

A doctor is not liable for: (1) **An error of judgement** or diagnosis, if he has secured all necessary data on which to base a sound judgement. (2) **Failure to cure or for bad results** that may follow, if he has exercised reasonable care and skill. Some risks are inherent in any form of treatment, and the doctor will not be negligent if they cause damage, provided that he has taken proper precautions, e.g., broken needle during injection. If the needle breaks, the patient should be informed and arrangements made to remove the broken piece. The doctor becomes negligent, if he fails to observe that the needle has broken, or having noted this, does not inform the patient or make arrangements to prevent further damage.

No doctor ensures success either in his diagnosis or in his treatment. There is always room for a difference of opinion among doctors. Bad results are not necessarily due to negligence, e.g., some patients may be keloid formers. **The law considers the doctor negligent only, when (1) he did not consider the possibility that such a complication might occur, (2) that he failed to watch for it carefully or to recognise it promptly, or (3) to treat in a timely and appropriate fashion.**

Degree of Competence: The doctors are expected to be well-informed of changing concepts and new developments and to follow general lines of treatment, though they are not expected to be aware of every development in medical science. **The degree of competence is not a fixed quality, but varies according to the status of the doctor.** A house surgeon is not expected to possess the same skills as a consultant surgeon, but he is expected to limit his activities (except in emergencies) to a level of medical care which is within his competence. **A general medical practitioner is expected to use only the average degree of skill and knowledge, possessed by doctors of similar status practising in the locality.** A specialist must maintain standards of skill in diagnosis and treatment above those of the ordinary general practitioner. **If a doctor claims to possess superior skill, knowledge, experience or training, he will be judged according to those standards even in its absence.**

Standard of care: The standard of care while assessing the practice as adopted, is judged in the light of knowledge available at the time of the incident, and not at the time of trial. Similarly, when the charge of negligence arises out of failure to use some particular equipment, the charge would fail, if the equipment was not generally available at that particular time, i.e., at the time of incident. The burden of proving negligence lies on the plaintiff (patient).

Liability for injury to third parties: A doctor may perform an examination at the request of a third party for sole use by third party, e.g. to determine eligibility for employment, evaluation of disability, insurance, drunkenness, etc., i.e. to examine the patient for non-therapeutic purposes. In this no physician-patient relationship is established. **The employed physician owes no duty to the examinee other than to avoid causing an injury,** but is under a duty to use reasonable care to avoid injury. Physician's duty is owed to his employer.

THE DOCTRINE OF RES IPSA LOQUITUR: Ordinarily, the professional negligence of a physician must be proved in the Court by the expert evidence of another physician. **The patient need not prove negligence in case where the rule of *"res ipsa loquitur"* applies, which means "the thing or fact speaks for itself". The patient has to merely state what according to him was the act of negligence.** **Conditions to be satisfied:** (1) that in the absence of negligence, the injury would not have occurred ordinarily; (2) that the doctor had exclusive control over the injury-producing instrument or treatment; (3) that the patient was not guilty of contributory negligence. **This enables the patient's lawyer to prove his case without medical evidence.**

Examples: (1) Prescribing an overdose of medicine producing ill-effects. (2) Failure to give anti-tetanic serum in cases of injury causing tetanus. (3) Burns from application of hot water bottles or from X-ray therapy. (4) Breaking of needles. (5) Failure to remove the swabs during operation, which may lead to complications or cause death. (6) Blood transfusion misadventure. (7) Loss of use of hand due to prolonged splinting. This doctrine is applied both to civil and criminal negligence. This doctrine is rarely used successfully by patients.

"CALCULATED RISK" CASES: The theory of the calculated risk doctrine is that *res ipsa loquitur* should not be applied when the injury complained is of a type that may occur even though reasonable care has been employed. This doctrine is an important defence to any doctor sued for professional negligence, who can produce expert evidence or statistics demonstrating that the accepted method of treatment he employed involved unavoidable risks.

DOCTRINE OF COMMON KNOWLEDGE: This doctrine is based on the assumption, that the issue of negligence in the particular case is not related to technical matters which are within the knowledge of the medical profession. It is a variant of *res ipsa loquitur. In res ipsa loquitur,* the patient need not produce evidence as to both the standard of care and specific act or omission. In doctrine of common knowledge, the patient must prove the causative (negligent) act or omission, but he need not produce evidence to establish the standard of care.

Composite Negligence: It occurs when a patient suffers from any injury due to negligent acts of more than one person without the negligence of the patient. In such case, patient may claim compensation from any one of negligent person. The defendant negligent person may claim contribution from other negligent persons.

Medical Maloccurrence: Medicine deals with human beings, and there are many biological variations, which cannot always be explained, expected or prepared for. **In some cases, in spite of good medical attention and care, an individual fails to respond properly. This is called medical maloccurrence.** Inevitable accident is an accident not avoidable by any such precautions, as a reasonable man can be expected to take, e.g., breaking of a needle during intramuscular injection due to sudden muscular spasm, or damage to the recurrent laryngeal nerve during thyroidectomy.

NOVUS ACTUS INTERVENIENS: A person is responsible not only for his actions, but also for the logical consequences of those actions. This principle applies to cases of assault and accidental injury. Sometimes, such a continuity of events is broken by an entirely new and unexpected happening due to negligence of some other person. In such cases, the assailant will not be fully responsible for the ultimate harm. If the doctor is negligent, which results in a deviation from the logical sequence of events, then the responsibility for the subsequent disability or death may pass from the original incident to the later negligent action, by the principle of **"*novus actus interveniens*" (an unrelated action intervening)**. Most of such interventions are of a medical nature, e.g., leaving of a swab or a surgical instrument in the abdomen after the repair of an internal injury; accidental substitution of poisonous drug for the therapeutic drug, etc. This plea is rarely accepted by the Courts.

CONTRIBUTORY NEGLIGENCE: Contributory negligence is any unreasonable conduct or absence of ordinary care, on the part of the patient (or on the part of his personal attendants), which combined with the doctor's negligence contributed to the injury as a direct, proximate cause, and without which the injury would not have occurred. These include (1) failure to give the doctor accurate medical history. If the patient provides incomplete or inadequate information, it could result in misdiagnosis,

mistreatment and harm, (2) failure to cooperate with his doctor in carrying out all reasonable and proper instructions, (3) refusal to take the suggested treatment, (4) leaving the hospital against the physician's advice, (5) failure to seek further medical assistance if symptoms persist. **As such, the doctor's negligence is not the direct, proximate (actual or legal) cause of the injury suffered by the patient.** If the negligence of the patient occurs at the same time with that of the doctor, it is a good defence for the doctor.

Liability of the doctor: The doctor cannot plead contributory negligence if he fails to give proper instructions. The extent of contributory negligence may vary and with it will vary the doctor's liability, from complete freedom from liability to a substantial liability for damages. **Normally, contributory negligence is only a partial defence,** and the Court has right to fix liability between the parties (doctrine of comparative negligence) and damages awarded may be reduced accordingly.

The burden of proof lies entirely on the doctor. If a doctor is not negligent, but if a patient is negligent which results in injury, it is called negligence of the patient.

Good Samaritan doctrine: One who assists another who is in serious danger cannot be charged with contributory negligence, unless the assistance is reckless or rash.

(2) CRIMINAL NEGLIGENCE: The **question** of criminal negligence may arise: (1) When a doctor shows gross absence of skill or care during treatment resulting in serious injury to or death of the patient, by acts of omission or commission. (2) When a doctor performs an illegal act. (3) When an assaulted person dies, the defence may attribute the death to the negligence in the treatment of the deceased by the doctor.

Conditions to be satisfied: Criminal negligence occurs if any one of the following are satisfied: (1) Indifference to an obvious risk of injury to health. (2) Actual foresight of the risk, but continuation of the same treatment. (3) Appreciation of the risk and intention to avoid it, but showing a high degree of negligence in the attempted avoidance. (4) Inattention or failure to avoid a serious risk.

Occurrence: Criminal negligence occurs when the doctor shows gross lack of competence or inaction, or inattention or gross recklessness, or wanton indifference to the patient's safety, gross negligence in the selection and application of remedies, or utter disregard for the life and safety of the patient, as to amount to a crime against the state, and conduct deserving punishment. **It involves an extreme departure from the ordinary standard of care. It is practically limited to cases in which the patient has died.** Most of such cases are associated with drunkenness or with impaired efficiency due to the use of drugs by doctors.

Examples: (1) Amputation of wrong finger or operation on wrong limb or wrong patient. (2) Leaving instruments, tubes, sponges or swabs in the abdomen. (3) Performing criminal abortion. (4) Administration of a wrong substance into the eye causing loss of vision. (5) Grossly incompetent administration of a general anaesthetic by a doctor addicted to the inhalation of anaesthetic. (6) Gross mismanagement of the delivery of woman, especially by a doctor under the influence of drink or drugs. Death resulting from an operation or injection of any drug producing anaphylaxis by a quack is considered criminal negligence.

Prosecution: Criminal negligence is more serious than the civil. **Section 304-A, I.P.C. deals with criminal negligence. "Whoever causes the death of any person, by doing any rash or negligent act not amounting to culpable homicide, shall be punished with imprisonment up to 2 years, or with fine or with both".** For criminal negligence, the doctor may be prosecuted by the police and charged in criminal Court, with having caused the death of the patient by a rash or negligent act not amounting to culpable homicide. **The prosecution must prove all the facts to establish civil negligence (except monetary loss), and gross negligence and disregard for the life and safety of the patient.** Contributory negligence is not a defence in criminal negligence (**Table 3-1**).

A physician may become liable to both civil and criminal negligence by a single professional act, e.g., if a physician performs an unauthorised operation on a patient, he may be sued in civil Court for damages and prosecuted in criminal Court for assault.

A doctor will not be criminally liable if a patient dies due to an error of judgement or carelessness or want of due caution though he can be liable to pay compensation.

Table (3-1). Difference between civil and criminal negligence

	Trait	Civil negligence	Criminal negligence
(1)	Offence:	No specific and clear violation of law need be proved.	Must have specifically violated a particular criminal law in question.
(2)	Negligence:	Simple absence of care and skill.	Gross negligence, inattention or lack of competency.
(3)	Conduct of physician:	Compared to a generally accepted simple standard of professional conduct.	Not compared to a single test.
(4)	Consent for act:	Good defence; cannot recover damages.	Not a defence; can be prosecuted.
(5)	Litigation:	Between two parties.	Between state and doctor.
(6)	Trial by:	Civil Court.	Criminal Court.
(7)	Evidence:	Strong evidence is sufficient.	Guilt should be proved beyond reasonable doubt.
(8)	Punishment:	Liable to pay damages.	Imprisonment.

Table (3-2). Difference between professional negligence and infamous conduct

	Trait	Professional negligence	Infamous conduct
(1)	Offence:	Absence of care and skill or wilful negligence.	Violation of Code of Medical Ethics.
(2)	Duty of care:	Should be present.	Need not be present.
(3)	Damage to person:	Should be present.	Need not be present.
(4)	Trial by:	Courts; civil or criminal.	State Medical Council.
(5)	Punishment:	Fine or imprisonment.	Erasure of name or warning.
(6)	Appeal:	To higher Court.	To State and Central Government.

CORPORATE NEGLIGENCE: The theory of corporate liability is typically applied in cases involving hospitals and their staff physicians. Hospitals have independent duty to their patients to investigate adequacy and review the competence of staff physicians. This theory is based on the principle that hospitals are in a far better position than their patients to supervise a physician's performance and provide quality control. This legal theory has been used to attack the allegedly negligent selection, retention or supervision of its participating physicians, that is negligent credentialing. It is the failure of those persons, who are responsible for providing the accommodation, facilities, and treatment to follow the established standard of conduct. It occurs when the hospital provides defective equipment or drugs, selects or retains incompetent employees, or fails in some other manner to meet the accepted standard of care, and such failure results in injury to a patient to whom the hospital owes a duty. In the corporate sector (hospital, nursing home, etc.), where more than one person, in more than one level, fails to render appropriate service to the patient, may result in some damage to patient. Here the treating doctor and other category of persons who were negligent and hospital administrators will be held responsible. If a hospital knows or should have known, that one of the patients is likely to be a victim of professional negligence by a doctor or its staff, the hospital is liable even though that doctor is an independent with staff privileges at the hospital. If the doctor is employed by a patient in his private capacity and the hospital only provides facilities for treatment, the doctor alone is held responsible for any negligence.

ETHICAL NEGLIGENCE: Ethical negligence is the violation of the Code of Medical Ethics. In this no financial compensation is payable, unless there is also civil negligence. If a complaint is made and the facts proved, the name of the doctor may be erased from the medical register. This term should be better avoided (Table 3-2).

Defensive medicine, i.e., ordering every test or X-ray on a patient to have a good background of hard data and avoiding using a potentially risky treatment which may offer much benefit to the patient. This may lead to withholding of beneficial treatment to the majority of patients because of a statistical risk to the

minority. The physician must not practice "defence by denial".

PRECAUTIONS AGAINST NEGLIGENCE: To prove that reasonable care and skill has been exercised, the following precautions should be taken: (1) Obtain informed consent of the patient. (2) Establish good rapport (relationship or communication) with the patient. (3) Keep full, accurate and legible medical records. (4) Employ ordinary skill and care at all times. (5) Confirm diagnosis by laboratory tests. (6) Take skiagrams in bone or joint injuries or when diagnosis is doubtful. (7) Immunisation should be done whenever necessary, particularly for tetanus. (8) Sensitivity tests should be done before injecting preparations which are likely to produce anaphylactic shock. (9) In suspected cases of cancer, all laboratory investigations should be done without delay to establish early diagnosis. (10) Seek consultation where necessary. (11) Do not criticise or condemn the professional ability of another doctor, especially in the presence of the patient. (12) Do not make a statement constituting or admitting fault on your part. (13) Avoid over-confident prognoses and promising too much to the patient. (14) Never guarantee a cure. (15) The patient must not be abandoned. (16) Inform the patient of any intended absence from practice or recommend or make available, a qualified substitute. (17) Transfer the patient if facilities are inadequate to handle his problem. (18) The drug should be identified before being injected or used otherwise. (19) Obtain consent for an operation or giving anaesthesia and to use discretion in obscure cases. (20) In a criminal wounding, operation should not be performed unless it is absolutely necessary. (21) Keep yourself informed of technical advances and use standard procedures of treatment. (22) No procedure should be undertaken beyond one's skill. (23) No experimental method should be adopted without the consent of the patient. (24) Frequently check the condition of equipment and use available safety installations. (25) Proper instructions should be given to the patient and proper postoperative care should be taken. (26) No female patient should be examined unless a third person is present. (27) Do not order a prescription over telephone, because of possibility of misunderstanding as to the drugs or their dosage. (28) Anaesthesia should be given by a qualified person. Only generally accepted anaesthesia should be given after clinical and laboratory examinations of the patient. (29) Exercise care in the selection of assistants and entrusting of duties to them. (30) Do not leave a patient unattended during labour.

Defences against negligence: (1) No duty owed to the plaintiff. (2) Duty discharged according to prevailing standards. (3) Misadventure. (4) Error of judgement. (5) Contributory negligence. (6) **Res judicata**, i.e., if a question of negligence against a doctor has already been decided by a Court, the patient cannot contest the same in another proceeding on the same set of facts. Only appeal can be made. (7) **Limitation:** A case should be filed within 2 years from the date of alleged negligence. Where breach of duty to provide care as per a contract between a patient and a doctor is committed, legal action can be initiated up to 3 years from the date of alleged negligence.

THERAPEUTIC MISADVENTURE: A **misadventure** is mischance, or accident or disaster. Misadventure is of three **types:** (1) **Therapeutic** (when treatment is being given). (2) **Diagnostic** (where diagnosis only is the objective at the time). (3) **Experimental** (where the patient has agreed to serve as a subject in an experimental study). **Therapeutic misadventure is a case in which an individual has been injured or had died, due to some unintentional act by a doctor or agent of the doctor or hospital.**

Almost every therapeutic drug and every therapeutic procedure can cause death. **A physician is not liable for injuries resulting from adverse reaction to drug, unless some negligence on his part contributed to cause the injury.** While prescribing a drug that has adverse side-effects, the doctor must be certain that the prescribed drug was the proper one for the disease. If there is any other drug which would be effective in treating the disease and is less likely to cause an allergic reaction, it should be prescribed. **Ignorance of the possibility of a reaction or continuation in the prescribing of a drug with adverse reaction amounts to negligence.**

Examples: (1) Hypersensitivity reaction, sometimes serious or fatal, may be caused by penicillin, aspirin, tetracycline, etc. (2) Prolonged

use of stilboestrol may cause breast cancer. (3) I^{131} therapy may cause thyroid cancer. (4) Electric equipment, hot water pads, and heating pads may produce burns. (5) Blood transfusion may cause serious or fatal complication from bleeding, resulting from haemolytic reaction due to hypofibrinogenaemia, and thrombocytopaenia. (6) Excessive administration of an antidote to a poisoned patient may cause death.

VICARIOUS LIABILITY: (Liability for act of another): An employer is responsible not only for his own negligence, but also for the negligence of his employees, if such acts occur in the course of the employment and within its scope, by the principle of *respondeat superior* (let the master answer).

Conditions to be satisfied: (1) There must be an employer–employee relationship, (2) the employee's conduct must occur within the scope of his employment, and (3) while on the job.

Examples: (1) In general practice, the principal doctor becomes responsible for any negligence of his assistant. Both may be sued by the patient, even though the principal has not been professionally concerned. The same applies where the principal employs non-medical servants. (2) When two doctors practise as partners, each is liable for negligence of the other, even though one may have no part in the negligent act. (3) When two or more independent doctors are attending on a patient, each may be held liable for the negligence of others that he observes, or in the ordinary course should have observed, and allows it to continue without objection. (4) **"Borrowed servant" doctrine:** An employee may serve more than one employer, e.g., the nurse employed by a hospital to assist in operations may be the borrowed servant of the operating surgeon during the operation, and the servant of the hospital for all other purposes. In this case, the lending employer temporarily surrenders control over his worker and the borrowing employer temporarily assumes control. If one doctor assists another in the operating room for a fee, the assistant is considered as an employee of the principal surgeon. (5) If a physician has supervisory control and the right to give order to a hospital employee in regard to the particular act and the employee is negligent in carrying out the act, the physician becomes legally liable for the harm caused by the employee. (6) If a swab, sponge, instrument, etc., is left in the patient's body after the operation, the surgeon is liable for damages. (7) A hospital as an employer is responsible for negligence of its employees who are acting under its discretion, supervision and control. It does not matter whether they are full-time or part-time, resident or visiting, permanent or temporary, because even if they are not servants, they are the agents of the hospital to give the treatment. (8) Hospital management will be held responsible for the mistakes of resident physicians and interns in training, who are considered employees when performing their normal duties. (9) A physician is responsible for the acts of the interns and residents carried out under his direct supervision and control. (10) Ordinarily, a surgeon is not liable for the negligence of anaesthetist, and the anaesthetist is not liable for the negligence of the operating surgeon. Physicians and surgeons are not responsible for the negligent acts of competent nurses or other hospital personnel unless such acts are carried out under their direct supervision and control. (11) When a doctor recommends another doctor to his patient after due care, he is not liable for the negligence of the new doctor, but he becomes liable if he knowingly refers his patient to an incompetent doctor. (12) Hospitals cannot be held responsible for the negligent acts of members of the superior medical staff in the treatment of patients, if it can be proved that the managers exercised 'the due care and skill' in selecting properly qualified and experienced staff. (13) If a physician has written a prescription properly, he is not liable for a pharmacist's negligence in preparing it, but he may be liable when he orders a prescription over the telephone resulting in misunderstanding as to the drugs or their dosage.

Liability: Usually, liability will be fixed upon those actually at fault and those whose control over the negligent is proved. The employer may be ordered by the Court to pay compensation to the injured patient. In such cases, the employer can engage in "third party proceeding" against the negligent doctor or employee asking for repayment.

PRODUCTS LIABILITY: Products liability refers to the physical agent which caused the injury or death of the patient during treatment by the doctor. The injury or death of the patient may result from the unexpected by-product of faulty, defective or

negligently designed medical or surgical instruments, or inadequate operating instructions. In such case, the manufacturer becomes responsible for injury or death.

Proof: **The doctor must prove that the manufacturer departed from standards of due care, with respect to negligent design, manufacture, assembly, packaging, failure to test and inspect for defects or failure to warn or give adequate instructions.** If the instrument functioned satisfactorily in previous operations or for several previous years in the hospital's possession, it is a proof that it was not defective at the time of supplying. If subsequently the instrument develops a defect through ordinary and gradual wear and tear, or if the physician or the hospital misuses the manufacturer's medical products, the hospital or physician owner are liable for the failure to inspect, test and repair such defects. But the manufacturer becomes responsible if the plaintiff can prove that the subsequent development of this defect was due to negligent design, structurally inferior component material, or improper assembly.

Drugs: The manufacturer of medicines has a legal duty to use care in research and development of drugs.

Liability: **The manufacturer is liable, if a patient is injured due to a drug reaction, negligence or breach of warranty. The manufacturer is also liable due to the harm caused by the contamination of a drug.** Products liability cases may be brought against manufacturers, sellers or anyone in the chain of sale. The plaintiff has to prove that a defect in the product existed before it left the manufacturer's hands and that the defect was the proximate cause of the plaintiff's harm.

MEDICAL INDEMNITY INSURANCE: **It is a contract under which the insurance company agrees, in exchange for the payment of premiums, to indemnify (reimburse to compensate) the doctor insured, as a result of his claimed professional negligence.**

Objects: (1) To look after and protect the professional interests of the insured doctor. (2) To arrange all other professional assistance including pre-litigation advice. (3) To arrange, conduct and pay for the defence of such doctors. (4) To indemnify the insured doctor arising from actions, claims and demands against him on grounds of professional negligence, misconduct, etc. When any dispute or allegation of negligence arises, the society must be contacted.

MEDICAL RECORDS: Objects: **(1) To serve as the basis for patient's care and for continuity in the evaluation of the patient's treatment. (2) To serve as documentation for reimbursement. (3) To provide data for use in medical education and clinical research. (4) To document communication between the doctor treating the patient and any other health care professional who contributes to the patient's care. (5) To assist in protecting the legal interests of the patient, the hospital and the practitioner responsible for the patient. (6) To follow up patients, evaluation of drug therapy and cost accounting.**

Requirements: The minimum requirements of accurate medical records are: (1) Evidence of informed consent. (2) An exhaustive history. (3) Relevant past and family history. (4) A description of the present disease or injury. (5) The report of physical examination showing objective findings and subjective complaint. (6) Diagnostic aids used and any reports received concerning the patient. (7) Impression of diagnosis. (8) Treatment, including medicines prescribed, and any procedures recommended or performed. (9) Complications if any. (10) Instructions given to the patient including diet. (11) Progress notes including clinical observations. (12) Notations concerning lack of co-operation by the patient. (13) Failure of the patient to follow advice or failure to keep appointments. (14) In emergency cases, specific clinical data and observations should be noted periodically. The omission of essential details from the notes may cast a doubt on the truthfulness of the witness.

Patient has the right to know what is in his records and is entitled to a copy of his hospital record on discharge, by paying the cost of reproduction. The next of kin can get the record in case of patient's death. Hospital has the responsibility to supervise the maintenance of appropriate, accurate, timely and up-to-date patient's records. The entries must be factual, and relevant. Nothing should be altered, deleted, substituted or added from the record, i.e., tampering should not be done. If tampering is done, patient may be awarded large sums, even though there has been no negligence. The police do not have a right to demand medical records except when there is statutory provision for such requisitions. The patient's record cannot be used in educational or diagnostic conferences or clinics or for publications, without the patient's consent. Hospitals have right to use

the medical records without the consent of patient for statistical purposes and quality of care determinations.

CONSENT IN MEDICAL PRACTICE

Consent means voluntary agreement, compliance or permission. To be legally valid, it must be given after understanding what it is given for, and of risks involved.

Reasons for obtaining Consent: (1) To examine, treat or operate upon a patient without consent is assault in law, even if it is beneficial and done in good faith. The patient may recover damages. (2) If a medical practitioner fails to give the required information to patient before asking for his consent to a particular operation or treatment, he may be charged for negligence.

Kinds of Consent: (1) **Express**, i.e. specifically stated by the patient. (2) **Implied**. **Express consent may be (a) verbal, or (b) written.**

Implied consent: The fact that a patient attends the hospital, or calls the doctor to his house complaining of illness, implies that he consents to a general physical examination, to determine the nature of the illness. Consent is implied when a patient holds out his arm for an injection. Such implied consent is the consent ordinarily given in routine practice.

Full Disclosure: The facts which a doctor must disclose depend on the normal practice in his community, and on the circumstances of the case. The doctor has to decide, after evaluating all aspects of the patient's personality, physical and mental state, how much can be safely disclosed. **A physician need not inform the patient of risks that a person of average intelligence would be aware of, or in an emergency situation. In general, the patient should ordinarily be told everything.**

Therapeutic privilege: This is an exception to the rule of "full disclosure". Full disclosure could result in frightening a patient who is already fearful, or who is an emotionally disturbed person, and who may refuse the treatment when there is really little risk. In these cases, the doctor may use discretion as to the facts which he discloses. The doctor should carefully note his decision in the patient's record explaining his intentions and the reasons. He should request a consultation to establish that the patient is emotionally disturbed. **If possible, the physician should explain the risks to the patient's spouse or next of kin.**

INFORMED CONSENT: Informed consent implies an understanding by the patient of (1) the nature of his condition, (2) the nature of the proposed treatment or procedure, (3) expectations of the recommended treatment and the likelihood of success, (4) the details of the alternative courses of treatment that are available, (5) the potential risks of not receiving treatment, (6) the risks and benefits involved in both the proposed and alternative procedure, (7) the relative chances of success or failure of both procedures, (8) particular known inherent risks that are material to the informed decision, so that he may accept or reject the procedure.

Exceptions to informed consent: (1) Emergency. (2) Therapeutic privilege. (3) When a patient waives his right to informed consent and delegates the right to the doctor or a close relative.

Prudent patient rule, i.e., what a prudent person in the patient's position would have decided if adequately informed about all the reasonably foreseeable risks.

Informed Refusal: The physician has a duty to disclose adequately and appropriately to the patient, the risks or possible consequences of refusal to undergo a test or treatment. After understanding all the facts, the patient can refuse to submit to treatment or an operation.

Paternalism: It is an abuse of medical knowledge so as to distort the doctor–patient relationship in such a way that the patient is deprived of his autonomy, or of his ability to make a rational choice, such as not disclosing the nature of illness and what is to be done, depriving the right of patient to accept or reject treatment.

RULES OF CONSENT: (1) **Consent is necessary for every medical examination.** Ordinarily, formal consent to medical examination is not required because the patient behaves in a manner which implies consent. (2) Oral consent should be obtained in the presence of a disinterested third party, e.g., nurse. (3) **Written consent should refer to one specific procedure, and not blanket permission on admission to hospital.** Written consent is not necessary in any case. However, it should be taken for proving the same in the Court if necessity arises. It should be witnessed by

another person. (4) **Any procedure beyond routine physical examination, such as operation, blood transfusion, collection of blood, etc., requires express consent.** It must be taken before the act but not at the time of admission into the hospital. Written consent should be in proper form and suitably drafted for the circumstances. The wording should include a phrase to confirm that the patient has been informed of the nature of the procedure. (5) The doctor should explain the object of the examination to the patient, and patient should be informed that the findings will be noted in a medical report. (6) **The doctor should inform the patient that he has right to refuse to submit to examination, and that the result may go against him. If he refuses it is an absolute bar for examination. (7) The consent should be free, voluntary, clear, intelligent, informed, direct and personal.** There should be no fraud, misrepresentation of facts, undue influence, compulsion and threat of physical injury, death or other consequences. (8) **In criminal cases, the victim cannot be examined without his/her consent.** The Court also cannot compel a person to get medically examined, against his will. (A) In cases of rape, the victim should not be examined without consent. (B) In medicolegal cases of pregnancy, delivery and abortion, the woman should not be examined without her consent. (9) A person may be arrested on a charge of committing an offence, and there may be reasons for believing that an examination of his person will provide evidence as to the commission of an offence. **A registered medical practitioner can examine an arrested person, even by using reasonable force, if the examination is requested by a police officer not below the rank of subinspector.** In the case of a female, the examination should be made only by or under the supervision of a female registered medical practitioner **(S.53 Cr.P.C.)**. "Examination" includes, examination of blood, bloodstains, semen, swabs in case of sexual offences, sputum, sweat, hair samples and fingernail clippings by the use of modern scientific techniques including DNA profiling and such other tests that a medical practitioner thinks necessary in that particular case. Under **S.54 Cr.P.C.** an arrested person at his request may be examined by a doctor to detect evidence in his favour. A copy of the report of such examination is to be furnished by the doctor to the arrested person or the person nominated by such arrested person. (10) In cases of drunkenness, the person should not be examined and blood, urine, or breath should not be collected without his written consent. But, if the person becomes unconscious or incapable of giving consent, examination and treatment can be carried out. The consent of guardian or of relatives, if available should be taken. **The person can be examined without consent, if requested by the subinspector of police.** (11) A person above 18 years of age can give valid consent to suffer any harm, which may result from an act, not intended or not known to cause death or grievous hurt **(S. 87, I.P.C)**. (12) A person can give valid consent to suffer any harm which may result from an act, not known to cause death, done in good faith and for its benefit **(S. 88, I.P.C)**. (13) A child under twelve years of age and an insane person cannot give valid consent to suffer any harm which may result from an act done in good faith and for its benefit. The consent of the parent or guardian should be taken **(S. 89, I.P.C.)**. If they refuse, the doctor cannot treat the patient even to save the life. **Loco parentis:** In an emergency involving children, if their parents or guardians are not available, consent is taken from the person-in-charge of the child, e.g., a school teacher can give consent for treating a child who becomes sick during a picnic away from home town, or the consent of the headmaster of a residential school. (14) A consent given by a person under fear of injury, or under a misconception of a fact is not valid. The consent given by an insane or intoxicated person, who is unable to understand the nature and consequences of that to which he gives his consent is invalid **(S.90, I.P.C.)**. (15) Any harm caused to a person in good faith, even without that person's consent is not an offence, if the circumstances are such that it is impossible for that person to signify consent, and has no guardian or other person in lawful charge of him from whom it is possible to obtain consent in time for the thing to be done in benefit **(S. 92, I.P.C.)**. (16) The doctor should inform reasonably to the patient about the nature, consequences and risks of the examination or operation before taking the consent to use his discretion. When there are two or more methods of treatment, the patient should be allowed to choose and give consent for any method. (17) The nature of illness

of a patient should not be disclosed to any third party without the consent of the patient. (18) **When an operation is made compulsory by law, e.g., vaccination, the law provides the consent.** (19) Consent of the inmates of the hostel, etc., is necessary if they are above twelve years. Within 12 years, headmaster or warden can give consent. (20) A prisoner can be treated forcibly without consent in the interest of the society. (21) **Consent given for committing a crime or an illegal act, such as criminal abortion is invalid.** (22) **Consent is not a defence in cases of professional negligence.** (23) For contraceptive sterilisation, consent of both the husband and wife should be obtained. (24) **The consent of one spouse is not necessary for an operation or treatment of other.** A husband has no right to refuse consent to any operation, including a gynaecological operation, which is required to safeguard the health of his wife. The consent of wife is enough. (25) It is unlawful to detain an adult patient in hospital against his will. (26) A living adult person can give consent for donating one of his kidneys to be grafted into another person. The donor must be informed of the procedure involved and possible risks. (27) If any person has donated his eyes to be used for therapeutic purpose after his death, **the eyes can be removed only with the consent of guardian or legal heirs.** (28) For organ transplantation, the organs of the dead person, such as heart, kidney, liver, etc., should not be removed without the consent of the guardian or legal heirs. (29) **Pathological autopsy should not be conducted without the consent of the guardian or legal heirs of the deceased.** If the autopsy is done without consent, the doctor is liable for damages for the mental anguish suffered by heirs, due to the mutilation of the body. Specific authorization should be obtained for retention of organs and parts of the body. **In medicolegal autopsies (statutory authorisation), consent is not required and the doctor can remove from the cadaver anything that is essential for purposes of examination.**

EUTHANASIA (mercy killing): It means causing painless death to a person suffering from hopelessly incurable and painful disease. **Active euthanasia** is an intentional, positive, merciful act, to end useless suffering or a meaningless existence. It is an act of commission, e.g., by giving large doses of drugs to hasten death. **Passive euthanasia** means discontinuing or not resorting to the use of extraordinary life-sustaining measures to prolong life in hopeless cases. This includes acts of omission, such as failure to resuscitate a terminally ill or hopelessly incapacitated patient or a severely defective newborn infant. **Voluntary euthanasia** means at the will of the person, and **involuntary** means against the will of the person, i.e., compulsory. **Non-voluntary** refers to cases of persons incapable of making their wishes known, e.g., in persons with irreversible coma or severely defective infants. Euthanasia advocates the administration of lethal doses of opium or other narcotic drugs. It has no legal sanction.

Netherlands was the first country to legalise euthanasia and assisted suicide in 2002, if the following conditions are met. (1) The disease is incurable. (2) The patient's suffering is unbearable. (3) Patient's condition is terminal. (4) Patient requests death.

Assisted Suicide: A person providing information to another with information, guidance and means to take his own life with the intention that it will be used for this purpose is assisted suicide.

Terminal sedation includes the administration of morphine and similar medications, which has a dual effect of relieving of pain and hastening the death **(aid-in-dying).** If the patient requests the same medical treatment with its known dual effect, and if the physician knowingly provides that medication by prescription so that patient can end his life, it is considered **physician-assisted suicide.**

MALINGERING: Malingering or shamming means conscious, planned feigning or pretending a disease for the sake of gain. The diseases that may be feigned are many, e.g., dyspepsia, intestinal colic, ulcers, spitting of blood, ophthalmia, diabetes, rheumatism, lumbago, neurasthenia, aphasia, sciatica, vertigo, epilepsy, insanity, paralysis of the limbs, burns, artificial bruises, etc. Patients can distort or exaggerate their symptoms, but true simulation is very rare. The history of the case should be taken from the person himself, and his relatives or friends and any inconsistencies in his description of the symptoms noted.

Diagnosis: **Usually the signs and symptoms do not conform to any known disease.** Malingering can be diagnosed by keeping the patient under observation and watching him without his knowledge. A thorough examination is essential after removing the bandages if any, and washing the part. Rarely, an anaesthetic may be given to detect malingering.

PENAL PROVISIONS APPLICABLE TO MEDICAL PRACTICE:

S. 52, I.P.C: Nothing is said to be done in good faith which is done without done care and attention.

S. 74, I.P.C.: Nonattendance in obedience to summons from court (six months imprisonment).

S. 118, I.P.C.: Concealing design to commit offence punishable with death or imprisonment for life. (imprisonment for three months if the offence is not committed and 7 years if the offence is committed).

S. 175, I.P.C.: Omission to produce document to public servant by person legally bound to produce it (six months imprisonment).

S. 176, I.P.C.: Omission to give notice or information to public servant by person legally bound to give it (imprisonment up to one month).

S. 177, I.P.C.: Furnishing false information (imprisonment up to six months).

S. 178, I.P.C.: Refusing oath or affirmation when duly required by public servant to make it (imprisonment up to six months).

S. 179, I.P.C.: Refusing to answer public servant authorised to question (imprisonment up to six months).

S. 182, I.P.C.: False information with intent to cause public servant to use his lawful power to the injury of another person (imprisonment up to six months)

S. 191, I.P.C.: Giving false evidence.

S. 192, I.P.C.: Fabricating false evidence.

S. 193, I.P.C.: Punishment for false evidence (imprisonment up to seven years).

S. 194, I.P.C.: Giving or fabricating false evidence with intent to procure conviction of capital offence (imprisonment up to ten years).

S. 195, I.P.C.: Giving or fabricating false evidence with intent to procure conviction of offence punishable with imprisonment for life imprisonment.

S. 197, I.P.C.: Issuing or signing false certificate (imprisonment up to seven years).

S. 201, I.P.C.: Causing disappearance of evidence of offence, or giving false information to screen offenders (imprisonment up to ten years).

S. 202, I.P.C.: Intentional omission to give information of offence by person bound to inform (imprisonment up to six months).

S. 203, I.P.C.: Giving false information respecting an offence committed (imprisonment up to two years).

S. 204, I.P.C.: Destruction of document to prevent its production as evidence (imprisonment up to two years).

S. 269, I.P.C.: Negligent act likely to spread infection of disease dangerous to life (imprisonment up to six months)

S. 270, I.P.C.: Malignant act likely to spread infection of disease dangerous to life (imprisonment up to two years).

S. 284, I.P.C.: Negligent conduct with respect to poisonous substances (imprisonment up to six months).

S. 39, Cr. P.C.: Every person, aware of the commission of, or of the intention of any other person to commit any offence punishable under I.P.C. shall forthwith give information to the nearest Magistrate or police officer of such commission or intention.

S. 160, Cr. P.C.: Police officer has the power to summon any witness (doctor) to police station for recording a statement.

S. 161, Cr. P.C.: The police has the power to examine witnesses.

S. 162, Cr. P.C.: Oral statements made to the police and recorded by the police should not be signed.

Legal protection to medical doctors is provided by S. 87 to 93, I.P.C.

CHAPTER

4 Identification

Identification is the determination of the individuality of a person based on certain physical characteristics, i.e., exact fixation of personality.

LEGAL ISSUES: Identification is necessary in: (1) living persons, (2) recently dead persons, (3) decomposed bodies, (4) mutilated bodies, and (5) skeleton. It is necessary in: (1) Criminal cases, such as persons accused of assault, murder, rape, etc., interchange of newborn babies in hospitals and in cases of impersonation. (2) In civil cases such as marriage, inheritance, passport, insurance claims, disputed sex, missing persons, etc. At least two identification marks should be noted by the doctor in all medicolegal cases. Before identifying the accused person in the Court, the doctor should verify the identification marks noted by him. Visual identification becomes difficult or impossible in cases of fires, explosions, advanced decomposition, mutilation, aircraft accidents, earthquakes, etc. Identification of a dead victim often enables the police to trace the victim's movements, his background, talk to his friends and to find out his enemies.

THE CORPUS DELICTI: The corpus delicti (delicti-fault; offence) (**the body of offence; the essence of crime) means, the elements of any criminal offence, e.g., murder.** The corpus delicti of homicide is the fact that a person died from unlawful violence. **It includes the body of the victim and other facts which are conclusive of death by foul play,** such as a bullet or a broken knife-blade found in the body and responsible for death. Clothing showing marks of the weapon and drawings and photographs of the deceased showing fatal injuries are also included in this term. **The main part of corpus delicti is the establishment of identity of the dead body and infliction of violence in a particular way, at a particular time and place, by the person or persons charged with a crime and none other.** The case against the accused cannot be established, unless there is convincing proof of these points. **The identification of a dead body and proof of corpus delicti is important before a sentence is passed in murder trials,** as unclaimed, decomposed bodies or portions of a dead body or bones are sometimes brought forward to support false charges. However, a conviction for an offence does not necessarily depend upon the corpus delicti, if there are eyewitnesses or strong corroborative evidence.

IDENTIFICATION DATA: (1) Race and religion. (2) Sex. (3) Age. (4) General development and stature. (5) Complexion and features. (6) External peculiarities such as moles, birth marks, malformations, scars, tattoo marks, wounds, occupation marks, etc. (7) Anthropometric measurements. (8) Fingerprints and footprints. (9) Teeth. (10) Personal effects: clothes, pocket contents, jewellery, etc. (11) Handwriting. (12) Speech and voice. (13) Gait, tricks of manner and habit. (14) Memory and education. 11 to 14 are useful only in the living. As no single feature is reliable for identification, a combination of features is taken. The primary characteristics of identification are: sex, age, and stature.

RACE

It can be determined by: (1) Complexion: The skin is brown in Indians, fair in Europeans and black in Negroes. (2) Eyes: Indians are dark-eyed, a few have brown eyes. Europeans have blue or grey eyes. (3) Hair: Indians have black, thin hair. Europeans fair or light-brown or reddish. Indians, Mongolians and Europeans have straight or wavy hair; Negroes have thick, short, curly hair. (4) Clothes: The dress may be helpful sometimes.

(5) **Skeleton:** The **Cephalic Index** (C.I.) or Index of Breadth is important.

$$\text{C.I.} = \frac{\text{Maximum breadth of skull}}{\text{Maximum length of skull}} \times 100$$

The length and breadth are measured by calipers, and not by measuring tape. The skull of an Indian is Caucasian with a few Negroid characters. Because of racial mixing, all the skulls may not be correctly differentiated into the three broad categories (**Table 4-1**).

RELIGION: Hindu males are not circumcised. Sacred thread, necklace of wooden beads, caste marks on forehead, tuft of hair on back of head and piercing

Table (4-1). Racial difference in the skull

Type of Skull	Cephalic Index	Race
Dolicho-cephalic (long-headed)	70 to 75	Pure Aryans, Aborigines and Negroes
Mesati-cephalic (medium-headed)	75 to 80	Europeans and Chinese
Brachy-cephalic (short-headed)	80 to 85	Mongolian

of ear lobes if present are helpful. Hindu females may have vermilion (cinnabar) on head, silver toe ornaments, *thali*, tattoo marks, nose-ring aperture in left nostril, few openings for ear-rings along the helix. Muslim females may have nose-ring aperture in septum only, several openings in the ears along helix and usually no tattoo marks. Muslim males are circumcised.

SEX

Sex has to be determined in cases of: (1) heir-ship, (2) marriage, (3) divorce, (4) legitimacy, (5) impotence, (6) rape, etc.

SEX CHROMATIN: It is a small planoconvex mass, about one micron in diameter **(Barr body)**, lying near nuclear membrane. In the buccal smear, the percentage of nuclei containing chromatin body ranges from zero to four in males and twenty to eighty in females **(Fig. 4-1)**. In females, neutrophil leucocytes contain a small nuclear appendage of drumstick form **(Davidson body)** in up to 6 per cent of cells. This is absent in males. In decomposed bodies, sex chromatin cannot be made out.

Sex can be determined from dental pulp, bone marrow and amniotic fluid. Karyotyping (study of chromosomal constitution of the nucleus of a cell for sex determination) of foetus may be done from lymphocytes, amniocytes and fibroblasts.

In normal cases, sex determination is easy from external examination only, but it is difficult in cases of (1) hermaphroditism, (2) advanced decomposition, and (3) skeleton.

INTERSEX: It is a condition in which characters of both sexes (in varying degrees) are present in one individual, including physical form, reproductive organs and sexual behaviour. It results from some defect in the embryonic development. It can be divided into four groups.

(1) Gonadal Agenesis: In this condition the testes or ovaries have never developed. The nuclear sex is negative.

(2) Gonadal Dysgenesis: In this condition, the external sexual structures are present, but at puberty the testes or the ovaries fail to develop.

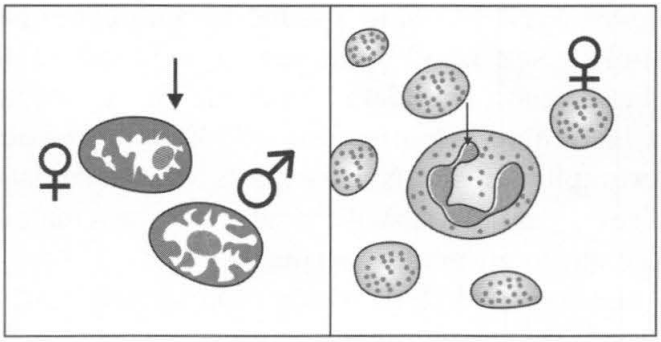

Fig. (4-1). The sex chromatin.

(A) KLINEFELTER'S SYNDROME: In this condition, the anatomical structure is male, but the nuclear sexing is female. The sex chromosome pattern is XXY (47 chromosomes). It is usually diagnosed when there is a delay in onset of puberty, behavioural disorders and mental retardation. Axillary and pubic hair are absent, and hair on chest and chin are reduced. Gynaecomastia, azoospermia, low levels of testosterone, sterility, increased urinary gonadotrophins, signs of eunuchoidism and increased height are common. Testicular atrophy with hyalinisation of seminiferous tubules is seen histologically.

(B) TURNER'S SYNDROME: In this condition, the anatomical structure is female, but the nuclear sexing is male. The sex chromosome pattern is XO (45 chromosomes). It can be recognised at birth by oedema of the dorsum of the hands and feet, loose skin folds in the nape of the neck, low birth weight and short stature. It is characterised by primary amenorrhoea, sterility, lack of development of primary and secondary sexual characteristics, increased urinary gonadotrophin excretion, pigmented naevi, a short fourth metatarsal, webbed neck, shield chest, wide-set nipples, high-arched palate, low-set ears, slow growth, learning problems, spina bifida, coarctation of aorta, septal defects, renal defects, Cushing's syndrome and a high incidence of diabetes mellitus. The ovaries do not contain primordial follicles (ovarian dysgenesis).

(3) True hermaphroditism: This is a very rare condition of **bisexuality in which an ovary and a testicle or two ovotestis are present with the external genitalia of both sexes.** There may be uterus. Phallus may be penile or clitoral; the labia may be bifid as in female or fused resembling the scrotum

of the male. Some develop female breasts and some menstruate. Neither gonad is completely functional. The somatic sex chromatin may be male or female.

(4) Pseudo-hermaphroditism: In this condition, the presence of **gonadal tissue of only one sex is seen internally, but external appearance is of the opposite sex.** (A) **Male pseudo-hermaphroditism:** Nuclear sex is XY, but sex organs and sexual characteristics deviate to female form, because of **testicular feminisation**. (B) **Female pseudo-hermaphroditism:** Nuclear sex is XX, but deviation of sex organs and sexual characters towards male are seen due to **adrenal hyperplasia**.

Persons with XYY chromosomal pattern are of aggressive and of criminal nature.

Concealed Sex: Criminals may conceal their sex to avoid detection by changing dress or by other methods. This can be detected by physical examination.

In advanced putrefaction, sex can be determined by identifying uterus or prostate, which resist putrefaction for a long time.

Skeleton: **Recognisable sex differences do not appear until after puberty except in the pelvis, and the accuracy from this bone is about 75 to 80% and without pelvis, only 50%.** Sex determination is rarely based on any one skeletal feature alone. **The determination of sex is based mainly upon the appearances of pelvis, skull, sternum and long bones. In the skull sexual features are modified by senility. In the pelvis, sex features are independent of each other and one may even contradict the other in the same pelvis. (Table 4-2 and Figs 4-2 to 4-11).** Perforated olecranon fossa is more common in females on the left side. According to Krogman, the degree of accuracy in sexing adult skeletal remains is:

Entire skeleton	100%
Pelvis alone	95%
Skull alone	90%
Pelvis plus skull	98%
Long bones alone	80%

Most anatomists do not claim 100% accuracy even where skeleton is available. One can expect 95% success rates.

In general adult female skeletal measurements are about 94% that of the male of the same race, but different measurements may vary from 91 to 98%.

Medullary Index of Bones: The sex of the long bones can be determined on the basis of their medullary index (diameter of medulla ÷ diameter of whole bone × 100) from tibia, humerus, ulna and radius. There is also a correlation between this index and the age of the subject.

AGE

The age of an individual especially in earlier years can be determined from: (1) teeth, (2) ossification of bones, (3) secondary sex characters, and (4) general development in case of children.

TEETH: At birth, the rudiments of all the temporary teeth and of the first permanent molars may be found in the jaws. Root formation begins after completion of the crown and as the root becomes longer, the crown erupts through the bone and finally comes out of the jaw bone. Neonatal line is well-formed on teeth at birth. During eruption of a permanent tooth, the overlying root of its deciduous predecessor simultaneously undergoes resorption, until only the crown remains. The unsupported crown is then shed.

Temporary Teeth: Temporary, deciduous or milk teeth are 20 in number: 4 incisors, 2 canines and 4 molars in each jaw. In ill-nourished children, hypothyroidism and especially in rickets, dentition may be delayed, while in syphilis teeth may be premature or even present at birth. **At about four years, there is usually spacing between the deciduous teeth. First permanent molar teeth erupt behind the second temporary molars about sixth or seventh year.** After this, the temporary teeth begin to be shed. **At the age of nine, 12 permanent teeth are in the mouth: 8 incisors and 4 first molars. Deciduous molars and canines should still be present. At the age ten, there are 16 permanent teeth (8 incisors, 4 premolars, 4 molars) and 8 deciduous (4 molars, 4 canines) teeth. At the age of eleven, there are 20 permanent teeth: 8 incisors, 8 premolars and 4 molars. Between 7 to 12 years, 24 teeth are present. At the age fourteen, there are 28 permanent teeth, and no deciduous teeth. If there is doubt whether a particular tooth is temporary, take an X-ray. If it is temporary, tooth germ of the permanent tooth can be seen underneath.**

Permanent teeth are 32 in number: 4 incisors, 2 canines, 4 premolars and 6 molars in each jaw. Developmentally, teeth are divided into two sets: **(1) Superadded permanent teeth are those which do not have deciduous predecessors. They erupt behind the temporary teeth. All the permanent molars are superadded permanent teeth. The first permanent molar tooth of each side erupts, while**

Table (4-2). Traits diagnostic of sex

	Trait	Male	Female
		GENERAL	
(1)	General size:	Larger, more massive.	Smaller, slender.
(2)	Long bones:	Ridges, depressions and processes more prominent. Bones of arms and legs are 8% longer.	Less prominent.
(3)	Shaft:	Rougher.	Smoother.
(4)	Articular surfaces:	Larger.	Smaller.
(5)	Metacarpal bones:	Longer and broader.	Shorter and narrower.
(6)	Weight:	4.5 kg.	2.75 kg.
		SKULL (Figs 4-3 and 4-8)	
(1)	General appearances:	Larger, longer (dolichocrania).	Smaller, lighter, walls thinner rounder and smoother (brachycrania).
(2)	Capacity:	1500 to 1550 ml.	1350 to 1400 ml.
(3)	Architecture:	Rugged; muscle ridges more marked, especially in occipital and temporal areas.	Smooth.
(4)	Forehead:	Steeper (sloping), less rounded.	Vertical, round, full, infantile.
(5)	Glabella:	Rough and more prominent.	Smooth, small or absent.
(6)	Frontonasal junction:	Distinct angulation.	Smoothly curved.
(7)	Orbits:	Square, set lower on the face; relatively smaller, rounded margins.	Rounded, higher, relatively larger, sharp margins.
(8)	Supraorbital ridges:	Prominent and rounded.	Less prominent, shorter or absent.
(9)	Cheek bones:	Heavier, laterally arched.	Lighter, more compressed.
(10)	Zygomatic arch:	More prominent.	Less prominent.
(11)	Nasal aperture:	Higher and narrower; margins sharp.	Lower and broader.
(12)	External auditory meatus:	Bony ridge along the upper border is prominent.	Often absent.
(13)	Frontal eminences:	Small.	Large.
(14)	Parietal eminences:	Small.	Large.
(15)	Frontal sinuses:	Much developed.	Less developed.
(16)	Occipital area:	Muscle lines and protuberance prominent and rounded.	Muscle lines and protuberance not prominent.
(17)	Mastoid process:	Wider, longer, round, blunt.	Narrow, short, smooth, pointed.
(18)	Base:	Sites of muscular insertions more marked.	Sites of muscular insertions less marked.
(19)	Digastric groove:	More deep.	Less deep.

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	Trait	Male	Female
(20)	Condylar facet:	Long and slender.	Short and broad.
(21)	Occipital condyles:	Large.	Small.
(22)	Palate:	Larger, broader, tends more to U-shape.	Small, tends more to parabola.
(23)	Foramina:	Larger.	Smaller.
(24)	Foramen magnum:	Relatively large and long.	Relatively small and round.
(25)	Teeth:	Larger.	Smaller.
		MANDIBLE (Fig. 4-2)	
(1)	General size:	Larger and thicker.	Smaller and thinner.
(2)	Chin:	Square (U-shaped).	Rounded.
(3)	Body height:	At symphysis greater.	At symphysis smaller.
(4)	Ascending ramus:	Greater breadth.	Smaller breadth.
(5)	Angle of body and ramus (Gonion):	Less obtuse (under 125°), prominent and everted.	More obtuse, not prominent, inverted.
(6)	Condyles:	Larger.	Smaller.
(7)	Mental tubercle:	Large and prominent.	Insignificant.
		PELVIS (Figs 4-4 to 4-10)	
(1)	Bony framework:	Massive, rougher, marked muscle sites. Stands higher and more erect.	Less massive, slender, smoother.
(2)	General:	Deep funnel.	Flat bowl.
(3)	Ilium:	Less vertical; curve of iliac crest reaches higher level and is more prominent.	More vertical; distance between iliac crests is less; iliac fossae shallow; curves of crest well-marked.
(4)	Preauricular sulcus:	Not frequent; narrow, shallow.	More frequent, broad and deep
(5)	Acetabulum:	Large, 52 mm in diameter; directed laterally, wider, deeper.	Small, 46 mm in diameter; directed anterolaterally; narrower.
(6)	Obturator foramen:	Large, often oval with base upwards.	Small, triangular with apex forwards.
(7)	Greater sciatic notch:	Smaller, narrower, deeper.	Larger, wider, shallower.
(8)	Ilio-pectineal line:	Well marked and rough.	Rounded and smooth.
(9)	Ischial tuberosity:	Inverted.	Everted; more widely separated.
(10)	Body of pubis:	Narrow, triangular, thick; ischial ramus short.	Broad, square; ischial ramus is lengthened and narrowed, pits (scars of parturition) on posterior surface if borne children.
(11)	Ramus of pubis:	It is like continuation of body of pubis.	Has a constricted or narrowed appearance and is short and thick.
(12)	Ischiopubic rami:	More everted; thicker and rougher.	Less everted; thinner and smoother.

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Trait	Male	Female
(13) Symphysis:	Higher, bigger and narrow in width. Margins of pubic arch everted.	Lower, wider and rounded. Margins of pubic arch not everted; distance between two pubic tubercles greater.
(14) Subpubic angle:	V-shaped, sharp angle 70 to 75°.	U-shaped, rounded, broader angle 90° to 100°.
(15) Pelvic brim or inlet:	Heart-shaped.	Circular or elliptical; more spacious, diameters longer.
(16) Pelvic cavity:	Conical and funnel-shaped.	Broad and round.
(17) Pelvic outlet:	Smaller.	Larger.
(18) Sacro-iliac articulation:	Large, extends to two-and-half to three vertebrae.	Small, oblique, extends to two to two-and-half vertebrae.
(19) Sacro-iliac joint surface:	Large and less sharply angulated.	L-shaped and elevated anteriorly.
(20) Sacrum:	Longer, narrower with more evenly distributed curvature; promontory well marked. Body of first sacral vertebra larger.	Shorter, wider, upper half almost straight, curve forward in lower half; promontory less marked. Body of first sacral. vertebra small.
(21) Coccyx:	Less movable.	More movable.
(22) Ischiopubic index: $\left(\frac{\text{Pubic length in mm}}{\text{Ischial length in mm}} \times 100\right)$	73 to 94	91 to 115
(23) Sciatic notch index: $\left(\frac{\text{Width of sciatic notch}}{\text{Depth of sciatic notch}} \times 100\right)$	4 to 5	5 to 6
(24) Sacral index: $\left(\frac{\text{Breadth of base of sacrum}}{\text{Anterior length of sacrum}} \times 100\right)$	112	116
(25) Pubic ramus ratio:	1 : 1	2 : 1 or greater
	THORAX	
(1) General:	Longer and narrower.	Shorter and wider.
(2) Sternum:	Body longer and more than twice the length of the manubrium; upper margin is in level with lower part of the body of second thoracic vertebra; breadth more. Length more than 149 mm. (Ashley's rule).	Shorter and less than twice the length of the manubrium; upper margin is in level with lower part of the body of third thoracic vertebra; breadth less. Length less than 149 mm. (Ashley's rule).
Sternal index: $\left(\frac{\text{Length of manubrium}}{\text{Length of body}} \times 100\right)$	46.2.	54.3.
(3) Ribs:	Thicker, lesser curvature and are less oblique.	Thinner; greater curvature and more oblique.
(4) Clavicle:	Longer (151 to 153 mm), broader, heavier, less curved.	Smaller (138 mm), narrower, lighter, more curved.

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Trait	Male	Female
	VERTEBRAL COLUMN	
(1) Atlas, breadth:	**7.4 to 9.9 cm. Mean 8.3 cm.**	**6.5 to 7.6 cm. Mean 7.2 cm.**
(2) Length of vertebral column:	**70 to 73 cm.**	**60 cm.**
(3) Corporobasal index of sacrum: $\left(\frac{\text{Breadth of first sacral vertebra}}{\text{Breadth of the base of sacrum}} \times 100\right)$	**45**	**40.5**
	FEMUR (Fig. 4-11)	
(1) Head:	**Larger and forms about two-thirds of a sphere. Vertical diameter more than 47 mm.**	**Smaller and forms less than two-thirds of a sphere. Vertical diameter less than 45 mm.**
(2) Neck:	**Obtuse angle with the shaft, about 125°.**	**Less obtuse angle with the shaft.**
(3) Bicondylar width:	**74 to 89 mm.**	**67 to 76 mm.**
	SCAPULA	
(1) Height:	**More than 157 mm.**	**Less than 144 mm.**
(2) Glenoid cavity:	**Height greater (39.2 mm).**	**Height less (32.6 mm).**

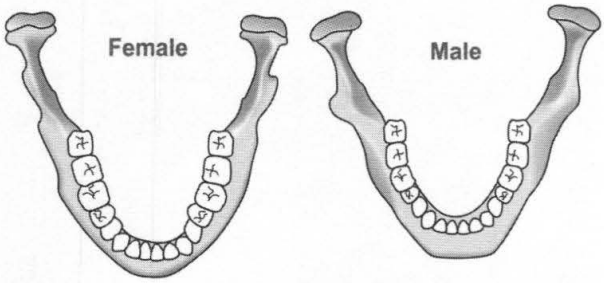


Fig. (4-2). Male and female mandibles.

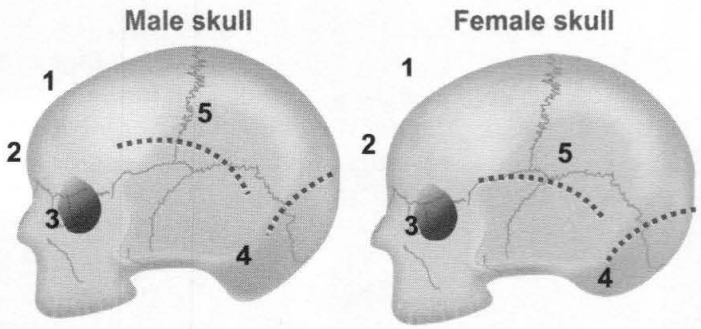


Fig. (4-3). MALE SKULL: (1) Forehead—receding. (2) Orbital ridges—prominent. (3) Orbits—square. (4) Mastoid process—large. (5) Muscle ridges—marked. **FEMALE SKULL:** (1) Forehead—high. (2) Orbital ridges—not marked. (3) Orbits—rounded. (4) Mastoid process—small. (5) Muscle ridges—faint.

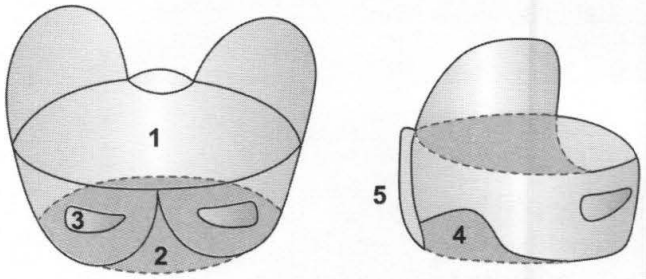


Fig. (4-4). FEMALE PELVIS: (1) General pelvic shape—flat bowl. (2) Subpelvic arch—obtuse. (3) Obturator foramen—oval. (4) Sciatic notch—obtuse. (5) Sacrum—short and flat.

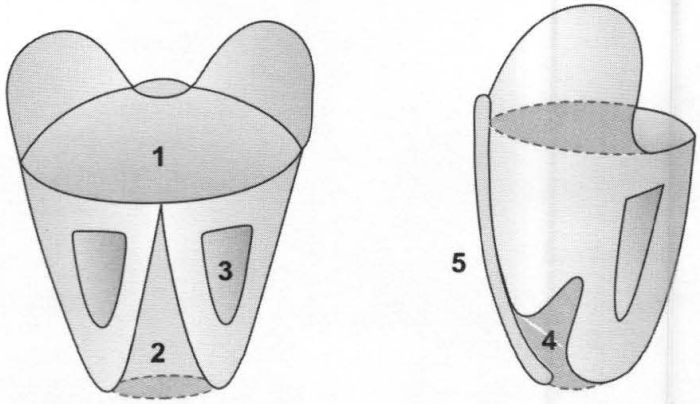


Fig. (4-5) MALE PELVIS: (1) General pelvic shape—deep funnel. (2) Subpelvic arch—acute. (3) Obturator foramen—triangular. (4) Sciatic notch—acute. (5) Sacrum—long and curved.

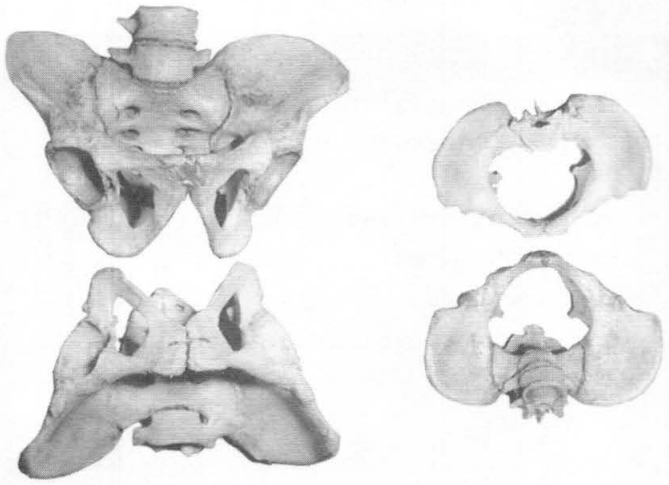


Fig. (4-6). Male (upper), female (lower) pelvis. The V-shaped subpubic angle and smaller pelvic outlet in male in contrast to U-shaped subpubic angle and larger pelvic outlet in female.

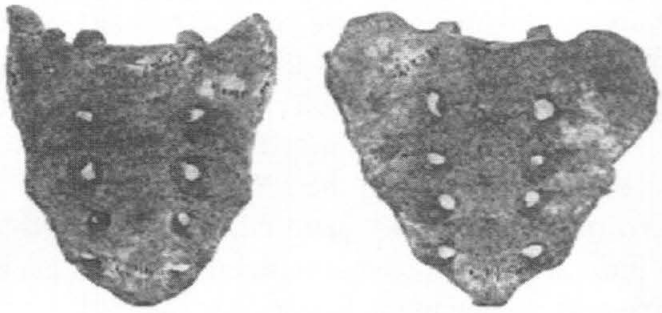


Fig. (4-7). Sacrum. It is narrower in the male (left) than in the female (right).

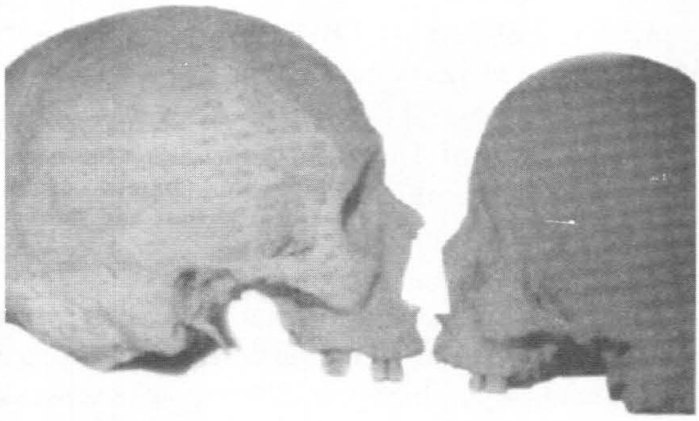


Fig. (4-8). Male (left) and female (right) skulls, showing more sloping forehead and more prominent mastoid process in the male in contrast to vertical, round forehead and less prominent mastoid process in female.

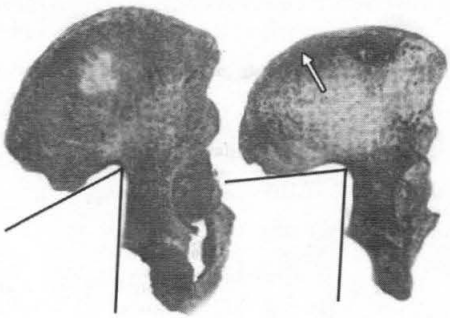


Fig. (4-9). Innominate bone. The angle formed by the greater sciatic notch is more acute in the male (left) than in the female (right).

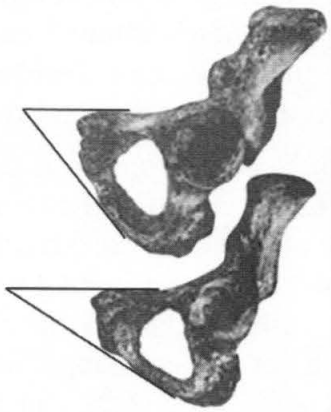


Fig. (4-10). Innominate bone. Male (above), female (below). Acetabulum is larger in the male. Body of pubis is narrow and triangular in male and broad and quadrangular in female. Inferior ramus of pubis has convex border in male and concave border in female.

Fig. (4-11). Male (right) and female (left) femurs. The angle of the neck of femur is more obtuse in the male.

Table (4-3). Difference between temporary and permanent teeth

	Trait	Temporary teeth	Permanent teeth
(1)	Size:	Smaller, lighter, narrower, except temporary molars which are longer than permanent premolars replacing them.	Heavier, stronger, broader, except permanent premolars replacing temporary molars.
(2)	Direction:	Anterior teeth are vertical.	Anterior teeth are usually inclined a little forward.
(3)	Crown:	China-white colour.	Ivory-white colour.
(4)	Neck:	More constricted.	Less constricted.
(5)	Root:	Roots of molars are smaller and more divergent.	Roots of molars are larger and less divergent.
(6)	Ridge:	A ridge or thick edge at the junction of the crown with the root present.	No ridge.

all the other deciduous teeth are present in the jaw. Superadded permanent teeth are six in each jaw. (2) Successional permanent teeth are those which erupt in place of deciduous teeth. Permanent premolars erupt in place of deciduous molars. Successional teeth are ten in each jaw (Table 4-3).

Eruption of Teeth: **Eruption is defined as the superior part of the crown of the tooth appearing level with the surface of the alveolar bone. In both deciduous and permanent teeth, dentition occurs earlier in lower jaw** except for the lateral incisors which erupt earlier in upper jaw. The lower permanent incisors, premolars and molars erupt about one year earlier than do the corresponding teeth in the upper jaw. Wisdom tooth first erupts in the lower and on the left side and then on the right side. **The number and eruption of deciduous teeth is more regular than the permanent dentition.** Eruption is not always bilaterally symmetrical. **Tooth eruption in females may be one year before that of males.** Females have smaller teeth (esp. lower canines) than males. In general, the **dental and skeletal ages correspond closely in the male, but in the female the skeletal age is generally one year ahead of the dental age.** Heredity, environment, endocrine reactions, and nutrition, all play a part in the eruption and calcification of teeth. Eruption tends to occur earlier in warmer climates and in urban areas **(Tables 4-3 to 4-5 and Figs 4-12 and 4-13).**

From birth to fourteen years of age, the degree of formation of root and crown structures, the stage of eruption and the intermixture of temporary and permanent teeth are useful in age estimation. Dental X-rays show the developmental status of unerupted teeth and the degree of root completion in erupted teeth. **From 14 to 20 years, dental age estimation is based upon the stage of development of the third molar and the root formation of the second molar. There is much variation in these, and the accuracy of dental age estimation during this period varies by about plus/minus three years.** The body of the jaw grows posteriorly, and the ramus is elongated after eruption of second molar teeth. If third molars are absent, it should be noted whether there is a space in the jaw behind the second molar teeth. **If third molars are fully erupted, it indicates that an individual is at least seventeen years of age.** In some persons due to inadequate jaw space, the third molars never erupt into the oral cavity, particularly the mandibular third molars. Such trapped teeth are known as **impacted teeth**. All the teeth can be visualized by single X-ray by dental panoramic tomograph (orthopantogram) **(Plate 1-1). It is generally accepted that in a child, estimation of age from teeth gives better results than skeleton.**

GUSTAFSON'S METHOD: **The age estimation of adult over 21 years depends on the physiologic age changes in each of the dental tissues (Fig. 4-14).**

(1) **Attrition:** Due to wear and tear from mastication, **occlusal (upper) surface of the teeth is destroyed gradually.** It depends on the functional use of teeth and also upon the hardness of the enamel.

(2) **Periodontosis: Regression of the gums and periodontal tissues surrounding the teeth takes place in advancing age,** which gradually exposes the necks and the adjacent part of roots, due to which the teeth become loose and fall off. There may be deposition of hardened debris which occurs gradually over a long period.

Table (4-4). Calcification and eruption of deciduous teeth

Tooth	Eruption	Resorption of root begins	Exfoliation
	months	year	years
Central incisor			
Lower:	6 to 8	4th	
Upper:	7 to 9	5th	6 to 7
Lateral incisor			
Lower:	7 to 9	5th	7 to 8
Upper:	10 to 12	5th	
First molar	12 to 14	6th	9 to 11
Canine	17 to 18	8th	9 to 12
Second molar	20 to 30 months	7th year	10 to 12 years

Table (4-5). Calcification and eruption of permanent teeth

Tooth	Eruption	Calcification complete
First molar	6 to 7 years	9 to 10 years
Central incisor	6 to 8 years	10 years
Lateral incisor	7 to 9 years	11 years
First bicuspid	9 to 11 years	12 to 13 years
Second bicuspid	10 to 12 years	12 to 14 years
Canine	11 to 12 years	12 to 13 years
Second molar	12 to 14 years	14 to 16 years
Third molar	17 to 25 years	18 to 25 years

(3) **Secondary dentine: It may develop from the walls within the pulp cavity,** and diminish the size of the cavity. **First it is deposited at the pulp chamber and gradually extends downwards to the apex and may completely fill the pulp cavity**. This is partly due to ageing, and partly due to pathological conditions like caries, and periodontosis.

(4) **Cementum apposition:** The cementum increases in thickness particularly due to changes in the tooth position, especially near the end of the root. **Secondary cementum is slowly and continuously deposited throughout life, and forms incremental lines which appear as cross-striations on the enamel, and are thought to represent daily increments of growth.**

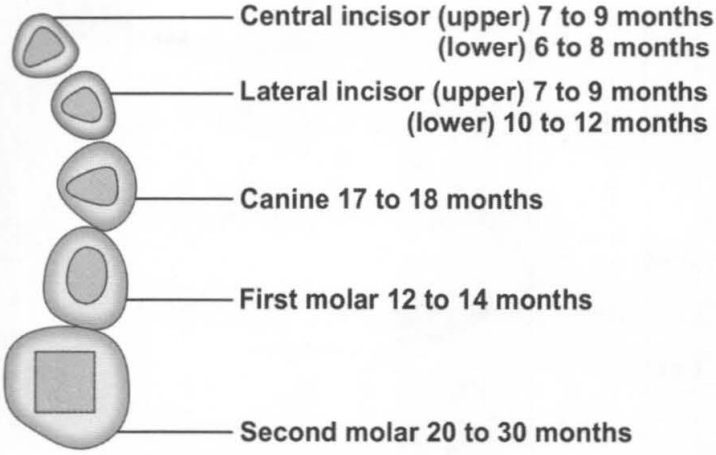

Central incisor (upper) 7 to 9 months
(lower) 6 to 8 months
Lateral incisor (upper) 7 to 9 months
(lower) 10 to 12 months
Canine 17 to 18 months
First molar 12 to 14 months
Second molar 20 to 30 months

Fig. (4-12). Eruption of temporary teeth.

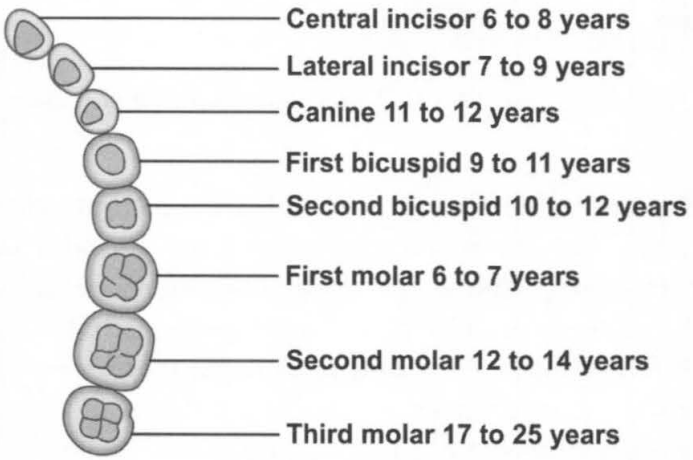

Central incisor 6 to 8 years
Lateral incisor 7 to 9 years
Canine 11 to 12 years
First bicuspid 9 to 11 years
Second bicuspid 10 to 12 years
First molar 6 to 7 years
Second molar 12 to 14 years
Third molar 17 to 25 years

Fig. (4-13). Eruption of permanent teeth.

(5) **Root resorption: It involves both cementum and dentine which show characteristically sharp grooves. Resorption of the root starts first at the apex and extends upwards.** It usually occurs in late age. It may be due to pathological process.

(6) **Transparency of the root: It is not seen until about thirty years of age.** The canals in the dentine are at first wide. With age they are filled by mineral so that they become translucent gradually and the dentine becomes transparent due to rarefaction (osteoporosis). **Transparency of root occurs from below upwards in lower jaw and from above downwards in upper jaw. It is the most reliable of all criteria.**

Before the tooth is extracted from a body, the degree of periodontosis is estimated. The tooth is rubbed on glass slabs from both sides of the tooth to about one mm which allows the estimation of transparency. Then the section is further rubbed to about one-fourth mm for microscopic examination.

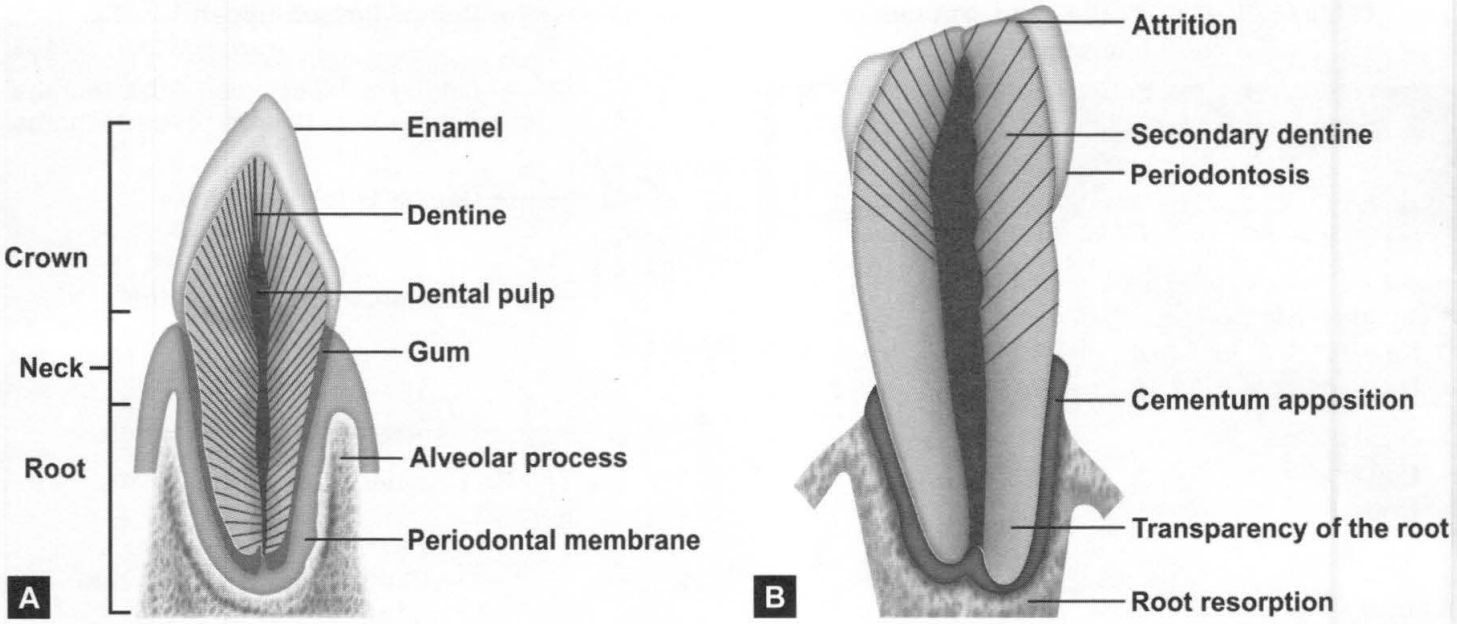


Fig. (4-14). (A) Vertical section of incisor tooth. (B) Physiological age processes in teeth.

Anterior teeth are more suitable than the posterior teeth, and the merit decreases from incisors to premolars, while third molar is quite unsuitable. All changes are absent at fifteen years. Arbitrarily, 0 to 3 points are allotted to indicate the degree of any of these changes. Stage 0 indicates no change, one beginning of change, two obvious change, and stage three maximum change, occurring in the dental tissues. The results corrected for standard deviation, etc., give an estimate of the age of the person. The error is said to be ± 4 to 7 years. The limit of error increases above 50 years of age.

Gustafson's formula: Age can also be determined by directly reading from the graph on which the regression line is plotted.

Boyde's method: Cross-striations develop in the enamel of teeth till the complete formation of enamel. They represent daily incremental lines. **The age of an individual can be calculated in terms of days by counting the number of lines from the neonatal line onwards. Neonatal line** is formed very soon after birth and can be seen in about three weeks or by electron microscopy within one to two days after birth. It is useful to estimate the age of a dead infant.

Stack's method: Stack evolved a method to know the **age of the infants from the weight and height of the erupting teeth of a child.** This method can be used on both deciduous and permanent teeth during their erupting phase.

GROWTH IN INDIVIDUAL BONES

(Ossification of bones): The bones of the human skeleton develop from a number of separate centres of ossification and growth. The appearance of such centres of ossification is spread over a long period of time. A large number appear in embryonic life, some appear much later in prenatal life, and others appear after birth **(Table 4-6). At the eleventh intrauterine week there are 806 centres of bone growth; at birth about 450; while the adult skeleton has 206 bones**. This shows that 600 centres of bone growth have disappeared, i.e., they have united with the adjacent centres to form the adult bone. **The process of appearance and union has a sequence and a time. As a rule, ossification begins centrally in an epiphysis and spreads peripherally as it gets bigger. At first it is entirely amorphous, rounded and pinhead sized. As it grows, it takes on the osteological details of the bone.** Some bones are ossified from a single centre, e.g., carpus and tarsus. **The ossification centres in carpal bones appear as follows: capitate 2 to 3 months, hamate first year, triquetrum 3, lunate 4, scaphoid and trapezoid 5, trapezium 6, and pisiform 11 years (Fig. 4-24). At the end of one year, two carpal bones are seen in X-ray of the wrist. Between 2 and six years, the number of carpal bones present on X-ray indicates the approximate age in years.** Most bones are ossified from several separate centres, one

Table (4-6). Appearance of centres of ossification and union of bones and epiphyses

Age	Appearance of centre of ossification	Union of bone and epiphyses
5th year:	Head of radius, trapezium, trapezoid, scaphoid.	Greater tubercle fuses with head of humerus.
6th year:	Lower end of ulna.	Rami of pubis and ischium unite.
6th to 7th year:	Medial epicondyle of the humerus.	
9th year:	Olecranon.	
9th to 11th year:	Trochlea of humerus.	
10th to 11th year:	Pisiform.	
11th year:	Lateral epicondyle of humerus.	
13th year:	Separate centres in triradiate cartilage of acetabulum.	
12th to 14th year:	Lesser trochanter of femur.	
14th year:	Crest of ilium; head and tubercles of ribs.	Medial epicondyle of humerus; lateral epicondyle with trochlea; patella complete.
15th year:	Acromion.	Coracoid with scapula; triradiate cartilage of acetabulum.
16th year:	Ischial tuberosity.	Lower end of humerus; olecranon to ulna; upper end of radius; metacarpals; proximal phalanges.
18th year:		Head of femur; lesser and greater trochanter of femur; acromion; lower end of ulna.
18th to 19th year:	Inner end of clavicle.	Lower end of femur; upper end of tibia and fibula; head of humerus; lower end of radius.
20th to 21st year:		Iliac crest; inner end of clavicle; ischial tuberosity.

of which appears near the middle of the future bone. Typically, a long bone, such as the tibia has become ossified throughout its shaft (diaphysis) at birth, whereas its two ends (epiphyses) are later ossified by secondary centres.

Epiphyseal Plate: A layer of hyaline cartilage (epiphyseal plate) persists between the diaphysis and epiphysis. **The bone increases in length at this epiphyseal plate or disc (growth plate or growth cartilage), until its final dimensions are attained.** The process of union of epiphysis and diaphysis is called fusion. **Union is a process, not an event. The union in the long bones is interpreted as non-united, uniting (¼, ½, ¾, etc.), recently united and united, depending on the stage of union (Table 4-6).**

For determining the age, skiagrams of the shoulder, elbow, wrist, hip, knee, ankle and pelvis should be taken in anteroposterior direction.

Variability: In biology, stability is the exception, variability is the rule, i.e., there is really no average. There is only a central tendency with a normal range of variability. The variability increases with age. **As a general rule, the ageing of bones is more precise with respect to the appearance of centres of ossification, than it is with respect to the union of epiphyses. As a general rule, the secondary centres of the limb bones that appear first are the last to fuse, whereas the late forming epiphyses reach union with their primary centres in a shorter time period.**

A study of various authorities shows, that there is a considerable variation, regarding the ages at which the various centres of ossification in the epiphyses fuse with their respective diaphyses. **In the long bones of upper limbs, the union occurs earlier at the elbow joint and later at the wrist. Head of the humerus is the last long bone epiphysis to unite. In the long bones of the lower limbs, the union occurs later at the knee joint and earlier at the hip and ankle joints.**

Figures 4-15 to 4-26 show the process of ossification in males. They only indicate an average.

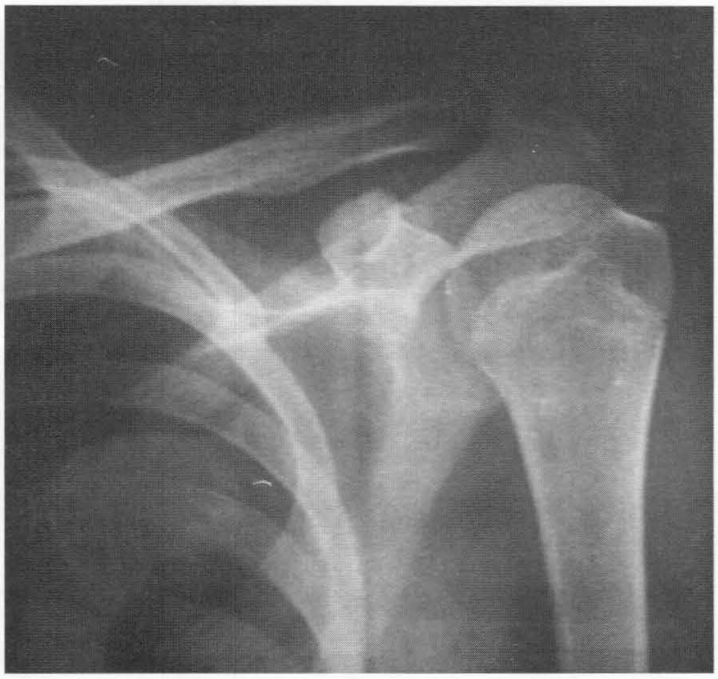


Fig. (4-15). X-ray photograph of the shoulder. The acromion, coracoid and head of the humerus not united with the shaft. Age 15 to 18 years.

Fig. (4-16). X-ray photograph of the knee. Fusion of the epiphyses of the lower end of femur and upper end of the tibia nearing completion. Age about 18 to 19 years.

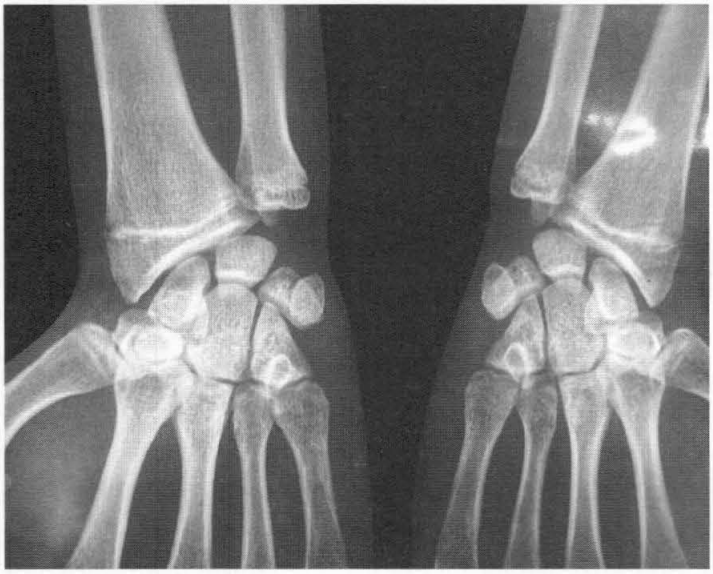


Fig. (4-17). X-ray photograph of the wrist and hand. All the carpal bones are seen. The lower epiphyses of first metacarpal not united with the shaft. Age between 12 to 15 years.

Fig. (4-18). X-ray photograph of the hip. Triradiate cartilage not obliterated. Greater trochanter of femur not united with upper end. Age below 14 years.

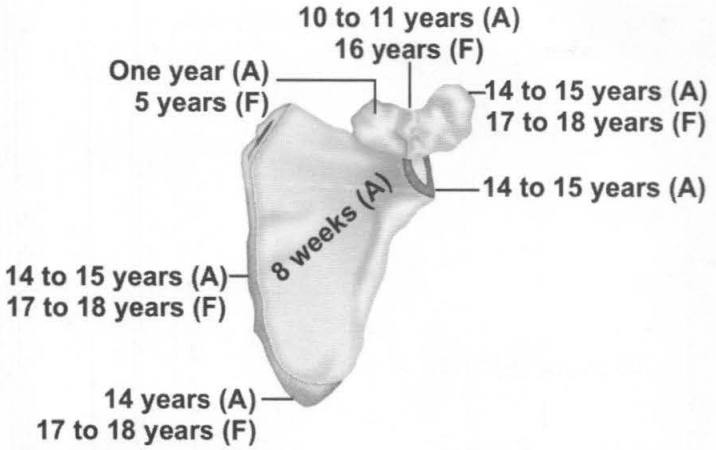

10 to 11 years (A)
16 years (F)
One year (A)
5 years (F)
14 to 15 years (A)
17 to 18 years (F)
14 to 15 years (A)
8 weeks (A)
14 to 15 years (A)
17 to 18 years (F)
14 years (A)
17 to 18 years (F)

Fig. (4-19). Appearance and fusion of epiphyses in scapula.

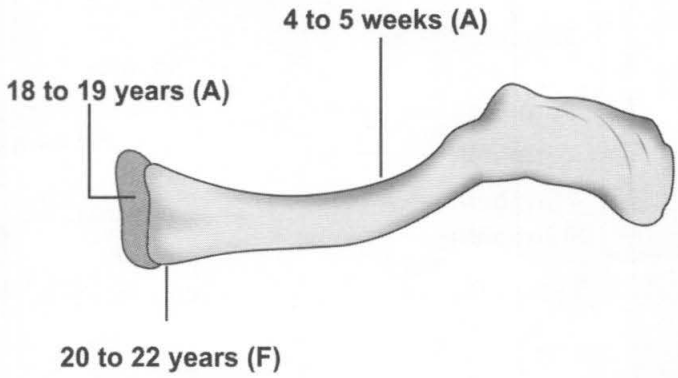


Fig. (4-20). Appearance and fusion of epiphyses in clavicle.

Too much reliance should not be placed on them, as variations occur depending on the health, hereditary, nutritional, endocrine and environmental factors. **Multiple criteria of skeletal age should be employed whenever possible. The dentition may be used as a check during the first two decades of life. An estimated skeletal age based on appearance of ossification centres and union of epiphyses must always be expressed in plus or minus terms, e.g., ten ± one. Skeletal development in the female can be in advance of the male up to one year, while dental development may differ only from one to four months. In males, dental and skeletal ages are almost similar, but in females osseous age is in advance of the dental age**. In tropical climates, ossification centres appear and epiphyseal union takes place about two years earlier than in temperate zones.

The epiphyseal lines on the long bones of a young individual appear as circular grooves around the ends of the bones, and on radiographs as irregular lines simulating a fracture. In skeletal remains of a young person, where the bones have become completely dry, the epiphyses often separate from the shaft, which should not be mistaken for fractures.

Union of epiphyses in cartilaginous bone occurs slightly earlier (by about one year) in the female than in the male, but the reverse is seen in the closure of the sutures of the skull.

Radiographs: The union of epiphyses as seen in radiographs appear earlier approximately about plus or minus six months, than the periods of fusion indicated by anatomical evidence. This is due to the fact that towards the end of the growth period, the epiphyseal plate of the cartilages becomes very thin and irregular in outline and may not show on radiograph. **In an individual bone, once union has begun, it will be completed in about 12 to 18 months.** In radiographs of growing long bones, one or more transverse lines are often observed at the diaphyseal ends. This is thought to be evidence of growth disturbance (e.g., scurvy, rickets), and are called "**scars of arrested growth**".

Hyoid Bone: The greater cornu of the hyoid bone unites with the body between 40 and 60 years.

Sternum: **The four pieces of the body of the sternum fuse with one another from below upwards between 14 and 25 years (Fig. 4-22)**. At about 40 years, the xiphoid unites with the body. The manubrium fuses with the body in old age (at about 60 years) **(Fig. 4-22)**.

Symphysis Pubis: The pubic symphysis is probably the best single criterion for determining age from third to fifth decades. The symphysial surface before twenty years has a layer of compact bone near its surface. At about 20 years, it is markedly irregular or uneven, and the ridges run transversely across the articular surface. Between 24 and 36 years, the ridges gradually disappear and the surface has a granular appearance, and ventral and dorsal margins are completely defined. Early in the fifth decade, the symphyseal face has an oval, smooth surface with raised upper and lower ends. Towards the end of the fifth decade, a narrow beaded rim develops on the margins. During the sixth decade, erosion of surface and breakdown of ventral margin begins. In the seventh decade, the surface becomes irregularly

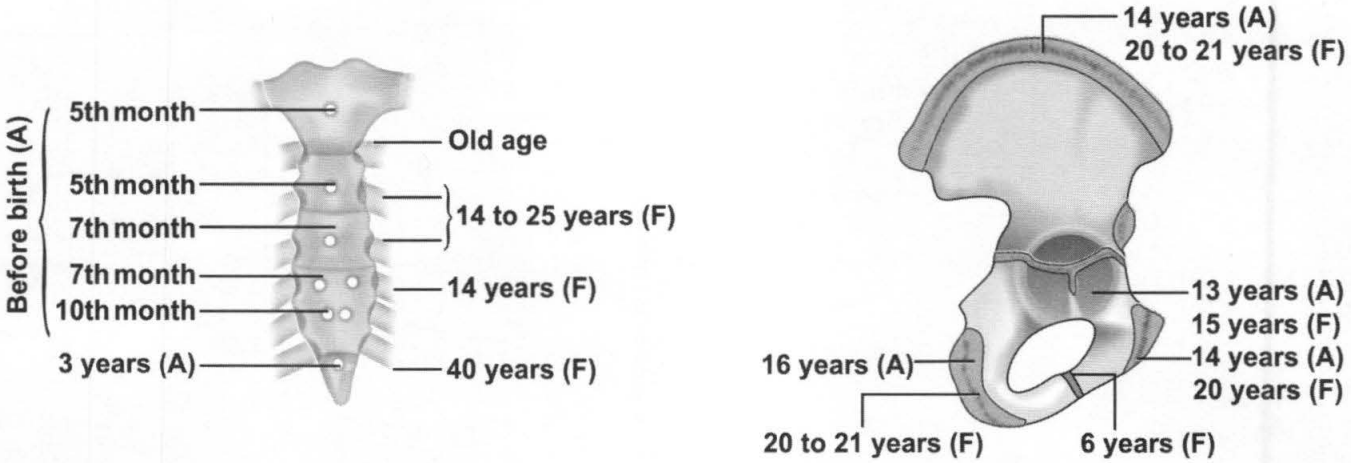


Fig. (4-21). Appearance and fusion of epiphyses in sternum and hip bone.

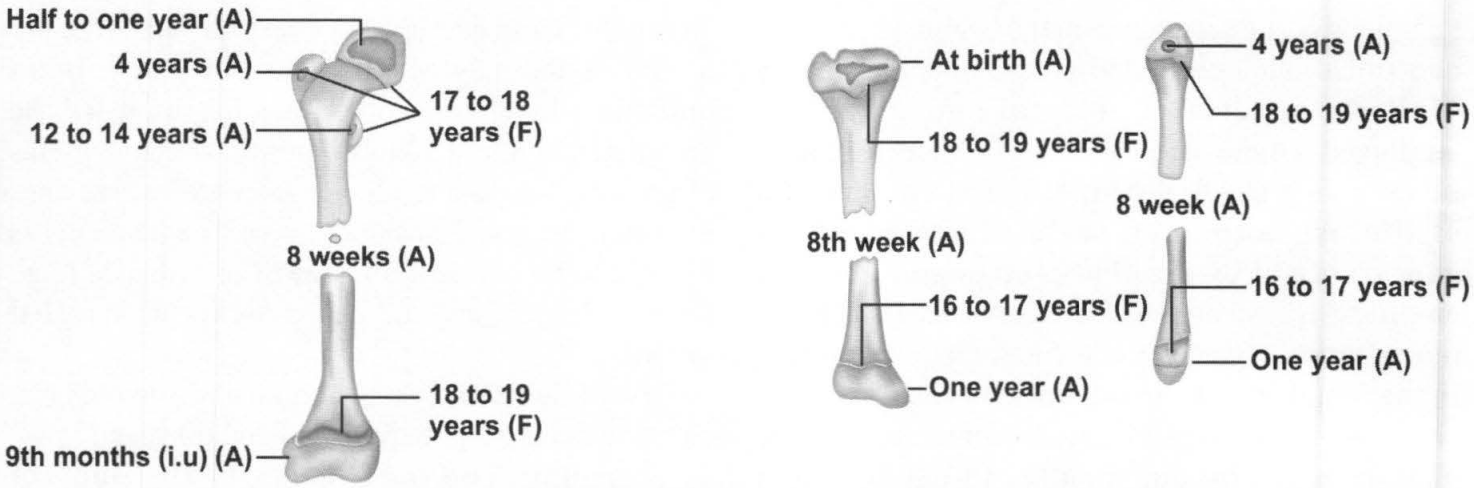

Half to one year (A)
4 years (A)
12 to 14 years (A)
17 to 18 years (F)
8 weeks (A)
18 to 19 years (F)
9th months (i.u) (A)
At birth (A)
18 to 19 years (F)
4 years (A)
18 to 19 years (F)
8 week (A)
8th week (A)
16 to 17 years (F)
One year (A)
16 to 17 years (F)
One year (A)

Fig. (4-22). Appearance and fusion of epiphyses in femur, tibia and fibula.

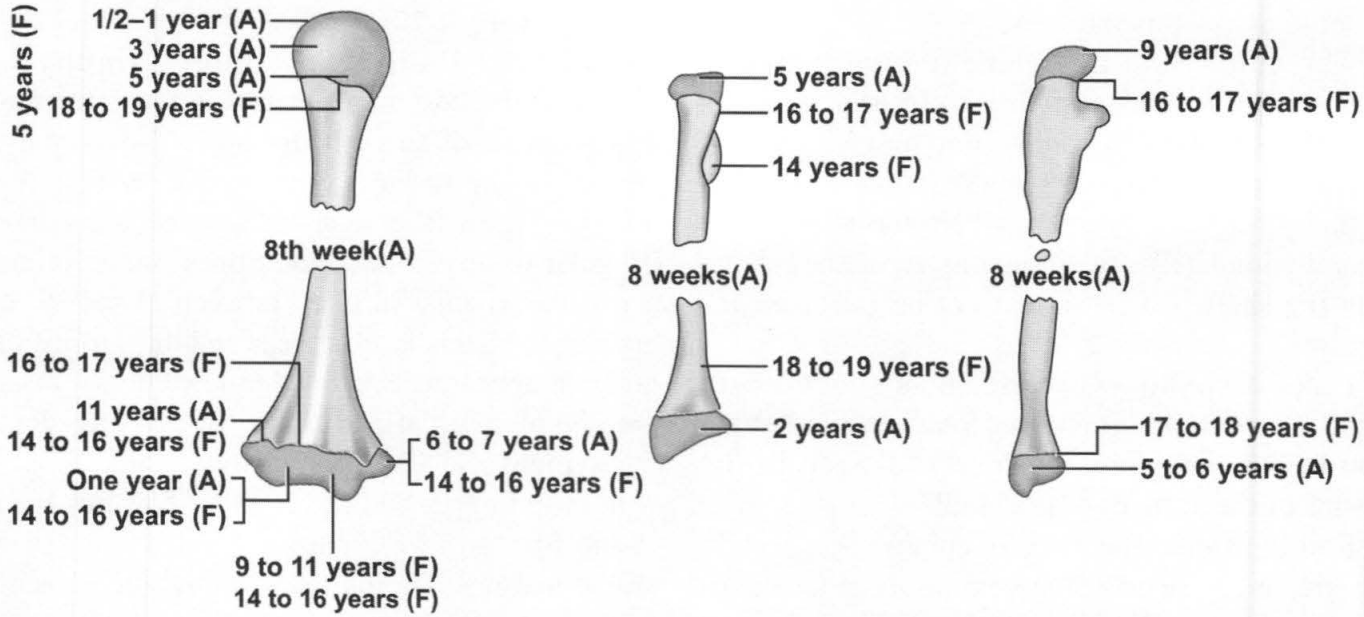


Fig. (4-23). Appearance and fusion of epiphyses in humerus, radius, and ulna.

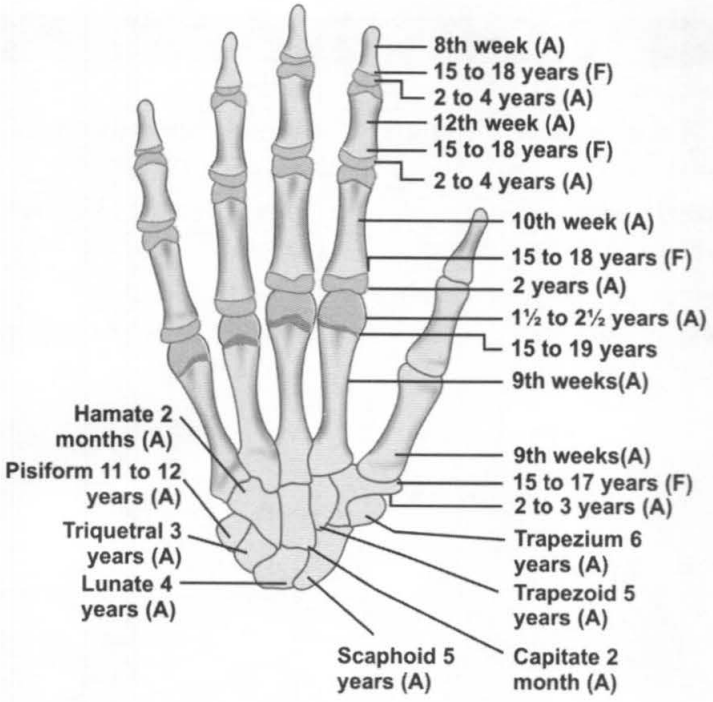

8th week (A)
15 to 18 years (F)
2 to 4 years (A)
12th week (A)
15 to 18 years (F)
2 to 4 years (A)
10th week (A)
15 to 18 years (F)
2 years (A)
1½ to 2½ years (A)
15 to 19 years
9th weeks(A)
Hamate 2 months (A)
Pisiform 11 to 12 years (A)
Triquetral 3 years (A)
Lunate 4 years (A)
9th weeks(A)
15 to 17 years (F)
2 to 3 years (A)
Trapezium 6 years (A)
Trapezoid 5 years (A)
Scaphoid 5 years (A)
Capitate 2 month (A)

Fig. (4-24). Appearance and fusion of epiphyses in bones of hand.

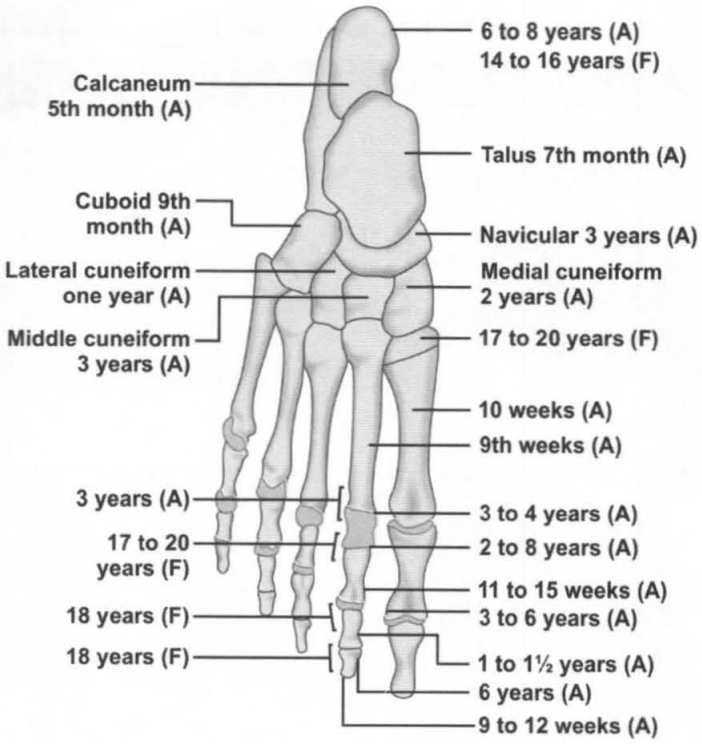

6 to 8 years (A)
14 to 16 years (F)
Calcaneum 5th month (A)
Talus 7th month (A)
Cuboid 9th month (A)
Navicular 3 years (A)
Lateral cuneiform one year (A)
Medial cuneiform 2 years (A)
Middle cuneiform 3 years (A)
17 to 20 years (F)
10 weeks (A)
9th weeks (A)
3 years (A)
3 to 4 years (A)
17 to 20 years (F)
2 to 8 years (A)
11 to 15 weeks (A)
18 years (F)
3 to 6 years (A)
18 years (F)
1 to 1½ years (A)
6 years (A)
9 to 12 weeks (A)

Fig. (4-25). Appearance and fusion of epiphyses in bones of foot.

eroded. If the male criteria is used for females, the age would be underestimated by about 10 years as the female pubis reaches full maturity about 10 years later than the male (**Fig. 4-27**).

SKULL: Bones of the calvaria are 8 in number: parietal 2, frontal 1, temporal 2, occipital one, sphenoid one, and ethmoid one. Bones of the face and jaws are 14 in number: maxilla 2, zygomatic 2, nasal 2, lacrimal 2, palatine 2, inferior nasal concha 2, mandible one and vomer one. The flexible cartilaginous joints of early life are replaced with interlocking connections between bones in maturity.

Suture closure: Posterior fontanelle closes in 6 to 8 months. The anterior fontanelle closes between 1.5 and 2 years and the two halves of the mandible unite at the second year. The condylar portions of occipital bone fuse with the squama at the third year, and with the basioccipital at the fifth year. The metopic suture closes about the third year, but in five to ten per cent cases it persists. The basioccipital fuses with the basisphenoid at about 18 to 21 years. In the vault of the skull, closure of the sutures begins on the inner side and proceeds externally. **The union occurs in the inner side of the skull 5 to 10 years earlier than on the outer side.** On the outer side, fusion occurs in the following order: posterior one-third of the sagittal suture at about 30 to 40 years;

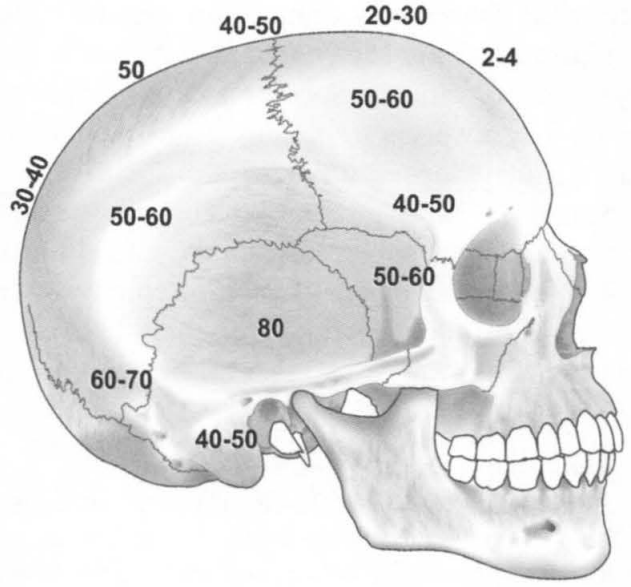


Fig. (4-26). Age of closure of cranial vault.

anterior one-third of the sagittal and lower half of the coronal at about 40 to 50 years; and middle sagittal and upper half of the coronal at about 50 to 60 years. The lambdoid suture starts closing near the lambda, and the union is often completed at about 45 years. The squamous part of the temporal bone usually fuses with its neighbour by the age of 60 years (**Fig. 4-26 and Plate 1-2**). **Suture closure in skull occurs later in females than in males. Estimation of age of skull from suture closure is not reliable. It**

Table (4-7). Difference between mandible in infancy, adult life and old age (Fig. 4-28)

Trait	Infancy	Adult	Old age
(1) Body:	Shallow.	Thick and long.	Shallow.
(2) Ramus:	Short and oblique; forms obtuse angle with body.	Less obtuse angle with the body.	Obtuse angle with the body, about 140°.
(3) Mental foramen:	Opens near the lower margin.	Opens midway between upper and lower margins.	Opens near the alveolar margin.
(4) Condyloid process:	At a lower level than coronoid process.	Elongated and projects above coronoid process.	At a lower level than coronoid process.

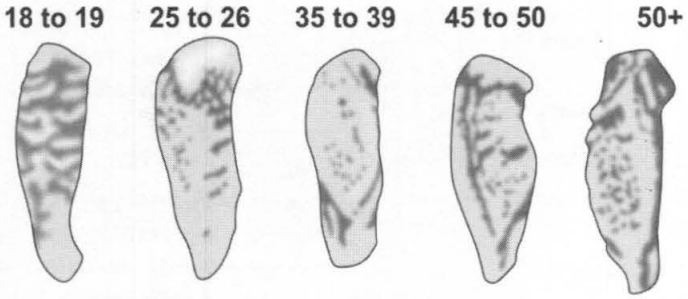


Fig. (4-27). Age changes in symphysis.

can be given only in a range of decade. The most successful estimate is done from sagittal suture, next lambdoid and then coronal. Ectocranial suture closure is very variable. Sometimes, ectocranial suture closure may not occur, especially in the sagittal suture. This is called **"lapsed union"**.

Sacrum: The five sacral vertebrae are separated by cartilage until puberty, when the lateral portions grow together. After this, fusion of epiphyses takes place and ossification of intervertebral discs extends from below upwards. The sacrum becomes a single bone between 21 and 25 years.

Mandible (Table 4-7 and Fig. 4-28):

SECONDARY SEX CHARACTERS: In the male, at about 14 years, fine hair begins to appear on pubis, the testes become large and firmer and the penis begins to enlarge. At about 15 years, hair is moderately grown on pubis, and hair begins to grow in axilla. At about 16 years, hair on pubis is well grown, and the private parts acquire a more adult appearance. Hair begins to make its appearance on the face between 16 and 18 years, and voice becomes hoarse.

In the female, the breasts begin to develop at about 13 years, the vulva becomes more horizontal due to forward tilting of the pelvis, the labia minora develop, and some fine pale, downy hair appears on mons veneris a couple of months later and menstruation starts. At about 14 to 15 years, the pubic hair is well grown and hair appears in the axilla.

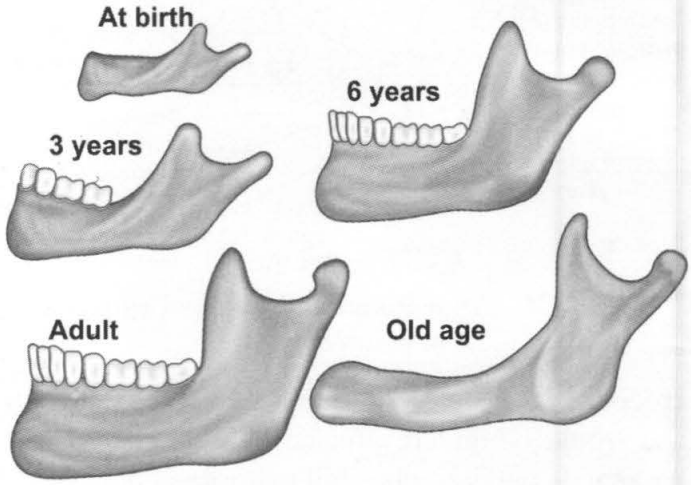


Fig. (4-28). The mandible at different periods of life.

CHANGES IN OLD AGE: BETWEEN 35 AND 50 YEARS, ossification of laryngeal and costal cartilages and hyoid bone usually start. Early changes in the articular surfaces of many bones may appear; they include lipping, reduction of joint space, and the presence of punched-out areas of osteoporosis on X-ray examination. The margins of the bodies of the lumbar vertebrae and the inner borders of the ischial tuberosities show lipping by 40 years, which become well advanced by 45 years. Wrinkles about the eyes, eyebrows and in front of ears appear about 35 to 40 years, and xiphoid process unites with the sternum at about 40 years. Greying of the hair is variable and not of much value. Head hair may become grey after 40 years, first at the temples. Later it involves the beard and still later chest, but pubic hair does not become grey before 50 to 55 years.

AFTER 50 YEARS: Joint changes become more extreme in character, and osteoporosis is more marked. The joint between the manubrium and the body of the sternum may fuse, and calcification of the laryngeal and costal cartilages becomes more visible. The hair may become silvery-white. Teeth are usually lost in old age. Loss of elasticity of skin, wrinkling and discolouration of the skin of buttocks and abdomen, atrophy of uterus and

ovaries, brown atrophy of the heart and atherosclerotic change in the arteries give an indication of old age.

Loss of collagenous stroma occurs in old age due to which the bone becomes lighter. The stroma is lost first in the outer cortex and the zone around the marrow cavity. The ends of long bones adjacent to the joints are the first to appear fragile and brittle. Radiological thinning of the cortex and progressive rarefaction of the apex of the medullary cavity in the head of the humerus and femur are helpful in the determination of age.

After the age of 25 years, the estimation of age becomes more uncertain. A careful examination of all factors, may enable the doctor to make a fair approximation to the decade within which a person may be.

ARCUS SENILIS: **It is a grey opaque ring surrounding the margin of the cornea due to lipid infiltration (degenerative changes) may appear about 50 years or later,** but is not complete before 60 years. The ring of opacity is separated from the limbus by a clear zone. When it occurs in young adults below 40 years due to hyperlipidaemia, it is called **arcus juvenilis**.

PROCEDURE FOR THE DETERMINATION OF AGE: **In medicolegal cases, the age of a person is determined by medical officer on requisition from the police officer or Magistrate. Written consent of the person should be taken. The following particulars should be noted. (1) Name, father's name, age alleged by the person, sex, occupation, and address. (2) Date, time and place of examination. (3) Name of the police constable accompanying the person. (4) Marks of identification. (5) Name of the nurse or attendant present at the medical examination. (6) Height, and weight. (7) General build and changes of puberty. (8) Dental examination findings. (9) Radiological examination of the bones.**

X-rays: **For determination of age between 6 and 12 years:** take X-rays of (1) Elbow joint. (2) Wrist. **6 years:** lower end of ulna (A); **6 to 7 years:** medial epicondyle of the humerus (A); **9 years:** Olecranon (A); **9 to 11 years:** trochlea of humerus (A). **10th to 11th years:** Pisiform (A); **12th Year:** lateral epicondyle of humerus (A) fill 9.8. **13 to 16 years:** X-rays of (1) Elbow. (2) Pelvis. **13th year:** Separate centres in triradiate cartilage of acetabulum (A); **12 to 14 years:** lesser trochanter of femur (A); **14th year:** Crest of ilium (A); Fusion of medial epicondyle of humerus and lateral epicondyle with trochlea. **15th year:** Fusion of triradiate cartilage of acetabulum. **16th year:** Ischial tuberosity (A); fusion of lower end of humerus; olecranon to ulna; upper end of radius.

Fusion of Bones/Joints:
16 years: elbow joint
16 to 17 years: ankle joint
17 to 18 years: hip joint
18 to 19 years: knee, shoulder and wrist joints
20 to 21 years: fusion of ischial tuberosity, iliac crest and inner end of clavicle

The opinion about the age should be given based on the findings of physical, dental and radiological examination. Multiple criteria of skeletal age should be used.

MEDICOLEGAL IMPORTANCE OF AGE

(1) Criminal Responsibility: On completion of five years, child may be held responsible for wrecking a train or endangering the safety of commuters of train. **Any act which is done by a child under seven years of age is not an offence (S. 82, I.P.C.).** A child between seven and twelve years is presumed to be capable of committing an offence, if he attained "sufficient maturity of understanding to judge the nature and consequences of his conduct on that occasion" **(S. 83, I.P.C.).**

(2) Judicial Punishment: According to the Juvenile Justice (Care and Protection of Children) Act, 2000, **"juvenile" means a boy or girl, who has not completed eighteenth year of age.** "Juvenile in conflict with law" would mean a juvenile alleged to have committed an offence and not completed 18 years of age on the date of commission of such an offence. When a juvenile commits an offence, the Court may direct that such person may be kept in any Juvenile Home or Special Home established by the Government, in which there are facilities for education, vocational training and rehabilitation, etc. **The period of stay in a Juvenile Home should not extend beyond the age of 18 years. No delinquent (offender) juvenile shall be sentenced to death or imprisonment.** Juveniles aged between 16-18 years, who commit heinous offences like rape or murder (punishable with imprisonment of 7 years or more) will be tried as adults. However they will be no death penalty or life term (2015 amendments).

(3) Rape: Sexual intercourse by a man with a girl under 16 years, even if she is his own wife, or with any other girl under 18 years even with her consent is rape (S. 375, I.P.C.).

(4) Kidnapping S.361 to 369, I.P.C.): Kidnapping means taking away a person by illegal means. It is an offence (a) to kidnap a child with the intention of taking dishonestly any movable property, if the age of child is under ten years, (b) to kidnap a minor from lawful guardianship, if the age of a boy is under 16 and that of girl under 18 years, (c) to kidnap a minor for purposes of maiming or begging, (d) to procure a girl for prostitution, if her age is under 18 years, and (e) to import into India from a foreign country a girl for purposes of illicit intercourse, if her age is less than 21 years.

(5) Employment: A child below 14 years cannot be employed for any type of work. A person completing 15 years (adolescent), is allowed to work in a factory as an adult, if a fitness certificate is issued by a certifying surgeon.

(6) Attainment of Majority: A person attains majority on the completion of 18 years, but when a person is under a guardian appointed by the Court, he attains majority on the completion of 21 years.

(7) Evidence: Competency to give evidence depends not on age but on understanding. **A child of any age can give evidence**, if the Court is satisfied that the child is truthful.

(8) Marriage Contract: A female under 18 years and a male under 21 years cannot contract marriage.

(9) Infanticide: The charge of infanticide cannot be sustained, if the infant can be proved under the age of seven months of intrauterine life.

(10) Criminal Abortion: A woman who has passed the childbearing age cannot be charged of procuring criminal abortion.

(11) Identification: An approximate age is important in any chain of identity data, e.g., when a few days old child is alleged to be the newborn child.

AGE OF THE FOETUS

The term **'developing ovum'** is used for the first 7 to 10 days after conception, i.e., until the implantation occurs. It is called an **'embryo'** from one week to the end of the second month, and later it is called **'foetus'**. It becomes an infant when it is completely born.

GESTATIONAL AGE can be determined from maturation of chorionic villi, foot length and ossification centres. By end of first month, eyes are seen as two dark spots and mouth as cleft. By end of second month, hands and feet are webbed and placenta begins to form. By end of third month, pupillary membrane, nails, neck and limbs are formed. By end of fourth month, sex can be recognised and meconium found in duodenum. By end of fifth month, skin is covered with vernix caseosa and hair appears on head. Meconium is found at beginning of large intestine. By end sixth month: Length 30 cm; weight 700 gm. Eyebrows and eyelashes appear. Skin is red and wrinkled and subcutaneous fat begins to be deposited. Vernix caseosa is present. Meconium in transverse colon.

End of seventh month: **Length 35 cm;** crown-rump length 23 cm; foot length 8 cm; **weight 0.9 to 1.2 kg**. Nails are thick. **Eyelids open and pupillary membrane disappears.** Skin is dusky-red, thick and fibrous. **Meconium is found in the entire large intestine. Testes are found at external inguinal ring.** Gallbladder contains bile and caecum is seen in the right iliac fossa. **Ossification centre is present in the talus.**

End of eighth month: Length 40 cm; weight 1.5 to 2 kg. Nails reach the tips of fingers. Scalp hair is thicker and 1.5 cm in length. Skin is not wrinkled. **Left testis is present in the scrotum.** Placenta weighs 500 gm. Ossification centre is present in lower segment of sacrum.

End of ninth month: Length 45 cm; weight 2.2 to 3 kg. Scalp hair is dark and four cm long; **meconium is seen at the end of large intestine. Scrotum is wrinkled and contains both testes.** Placenta weighs 500 gm. Ossification centres are usually present in the lower end of the femur.

End of tenth month (full term child): Length 50 to 53 cm; crown-rump length 30 to 33 cm; **weight 2.5 to 5 kg; average about 3.4 kg. The length is much less variable than the weight.** The male infant weighs about 100 gm more than the female. The circumference of the head is 33 to 38 cm. **Anterior fontanelle is 4 cm × 2.5 cm. The surface of the brain shows convolutions, and the grey matter begins to form.** The scalp hair is dark, 3 to 5 cm long. Lanugo is absent except on the shoulders. **The face is not wrinkled.** The skin is pale and covered with vernix caseosa. The nails project beyond the end of fingers, but reach only the tip of the toes. **The cartilages have formed in the nose and ears. The testes are present in the scrotum; vulva is closed** and labia minora are covered by fully developed labia majora. The rectum

contains dark brownish, green or black meconium. **The umbilicus is situated midway between pubis and xiphoid cartilage. The centre of ossification is found in the lower end of femur, and sometimes in the cuboid and in the upper end of the tibia.** The placenta is 22 cm in diameter, one-and-half cm thick at the centre and weighs about 500 gm. The umbilical cord is 50 to 55 cm long and one cm thick.

Ossification Centres: (1) Sternum: It is placed flat on a wooden board and cut with the cartilage knife in its long axis in midline.

(2) Lower end of femur and upper end of tibia: The leg is flexed against the thigh and a transverse or vertical incision is made into the knee joint. The patella is removed. The end of the femur is pushed forward through the wound, and a number of parallel cross-sections are made through the epiphysis, starting from its articular surface and continuing until the largest part of ossific centre is reached. **The centre is seen as a brownish-red nucleus surrounded by bluish-white cartilage.** Further sections are made through plain cartilage above it, until the diaphyseal centre is reached. **The centre appears about the thirty-six week.** Its diameter is about 4 to 5 mm at 37 to 38 weeks and 6 to 8 mm at full term. The upper end of tibia is similarly examined. **In 80% of full term infants, a centre is present in the upper end of tibia but in other cases it appears after birth.**

(3) Bones of the foot: The foot is grasped in the left hand behind the heel, the toes pointing towards the dissector. An incision is made between the interspace of third and fourth toes with a long knife, backwards through the sole of the foot and heel. If centre in calcaneum and talus are not exposed, thin slices of cartilage of these should be cut, until the presence or absence has been shown. **Centre in the calcaneum appears at the end of the fifth, and in talus at the end of seventh month of intrauterine life.** A centre may be present in cuboid at birth or it may appear shortly afterwards.

RULE OF HAASE: This is a rough method of calculating the age of the foetus. The length of the foetus is measured from the crown to the heel in centimetres. **During the first 5 months of pregnancy, the square root of the length gives the approximate age of the foetus in months, e.g., a foetus of 16 cm is 4 months. Haase's modification of Morrison's law: During the last five months, the length in centimetres divided by five gives the age in months, e.g., foetus of 35 cm is 7 months.**

After birth, the length of an infant is 50 cm; 60 cm at the end of six months; 68 cm at the end of first year, and one metre (double its length at birth) at the end of fourth year. Birth weight doubles by about 5 months of age and triples by about one year. Head circumference increases by about 12 cm in the first year of life.

STATURE

Variations in Stature: Stature varies at different times of the day by one-and-half to two cm. It is less in the afternoon and evening due to the reduced elasticity of the intervertebral discs and the longitudinal vertebral muscles. Both malnutrition and advancing years reduce stature. **After the age of thirty**, the natural processes of senile degeneration (atrophic changes in disc cartilages or bones) cause **gradual decrease in stature by about 0.6 mm per year on an average. The stature is greater by one to three cm on lying.** On an average, the body lengthens after death by about one-and-half cm in the male and two cm in the female.

Determination of Stature: If the body has been dismembered, the approximate stature may be determined by measurement of the trunk or limbs. (1) The length from the tip of the middle finger to the tip of the opposite middle finger, when arms are fully extended closely equals the height. (2) Twice the length of one arm, with thirty cm added for two clavicles and four cm for the sternum, equals the height. (3) The length of forearm measured from tip of olecranon process to tip of the middle finger is equal to 5/19 of the stature. (4) The length from the vertex to the symphysis pubis is roughly half of stature. After 14 years of age the symphysis pubis lies about halfway up the body. Before 14 years, the trunk is longer than the lower limbs. (5) The height of head measured by the vertical distance from the top of the head to the tip of the chin is about one-seventh and the length of the skull is about one-eighth of the total height. (6) The length from the sternal notch to symphysis pubis multiplied by 3.3 gives the stature. (7) The length of vertebral column is 35% of the height. (8) To the length of entire skeleton add two-and-half cm to four cm for the thickness of the soft parts. (9) Maximum foot length divided by 0.15 gives stature.

ANTHROPOMETRY (Bertillon system): This is applicable only to adults. This system includes: (1) Descriptive data: Such as, colour of hair, eyes, complexion, shape of nose, ears, chin, etc. (2) Body marks: moles, scars, tattoo marks. (3) Body measurements: height, anteroposterior diameter of head and trunk, the span of outstretched arms, the length of left middle finger, left little finger, left forearm, left foot, length and breadth of right ear and colour of left iris. (4) The photographs of a front view of the head and a profile view of the right side of head are also taken. As a sole means of identification, photographs are not always reliable, and they may be a source of error even when they are inspected by experts. This system has now been replaced by dactylography.

SKULL-PHOTO SUPERIMPOSITION

Superimposition is the technique applied to determine whether the skull is that of the person in the photograph. It is based on the principle that the shape of the face is related to the bony contours of the facial part of skin. The photograph of the head and face is enlarged to natural size, from the presence of some standard things in the photograph of the missing person to indicate the scale. To find out the life size of the face, objects like spectacles, patterns in clothes, etc., seen in the original photograph should be available as reference. In the absence of reference object, life-size enlargement is made by taking the interpupillary distance (midpoint of the orbital cavities) as a reference measurement. The skull and mandible are mounted on a stand giving appropriate tilt similar to the tilt of the face in the photograph and a photograph taken with scale. **A negative is prepared by recopying it, and enlarged to life size using the scale as a reference object. The negatives of the photographs and the skull are superimposed by aligning the characteristic points in the negatives. The two superimposed negatives are then photographed on bromide paper.** The resulting superimposed photograph brings out the points of similarity or dissimilarity between the photograph and the skull. **The superimposition is correct if the anatomical landmarks, such as nasion, menton, gonion, prosthion, nasal spine, zygomas, supraorbital ridges, external auditory meatus, angles of the jaw and teeth and the size of the skull accurately correspond to the face in the photograph. This test is of a more negative value, because it can definitely be stated that the skull and the photograph are not those of the same person.** If they tally, it can only be stated that the skull could be that of the person in the photograph, because of the possibility that another skull of that size and contour may tally with the photograph.

VIDEO SUPERIMPOSITION: The skull is fixed on a rotatable universal stand. The photograph is also fixed with the same orientation parallel to the skull. One camera is directed at the skull and a second camera directed at the photograph of the face. Images of the skull and photographs are projected on a monitor placed near the skull. By performing a series of blending, fading and sweeping (vertical, horizontal and diagonal), the image of the skull and photograph are superimposed and analysed for conformity. All the anatomical landmarks are compared. This is an exclusionary method.

COMPUTER-ASSISTED SUPERIMPOSITION: The skull and facial photograph are digitised using a computer. The two images are compared morphologically, by image processing for conformity. All the anatomical landmarks are compared.

COMPUTER PICTURES: Several curves for each of facial creases, prominences, shape of forehead, eyes, ears, nose, cheek, lips, teeth projections, scalp hair, moustache and beard are stored in a computer. Depending on the descriptions of the different features by the persons who have seen the criminal, hundreds of varieties of face can be drawn on the screen of the computer within a few minutes. An almost exact appearance of the face can be drawn by additions and alterations of curves, which can be printed out. A major problem is that features which give individuality to a face, such as the eyes, lips and facial hair, are not very dependent upon the underlying bone structure.

PHYSIOGNOMIC RESTORATION: (1) Sculptural to give a three-dimensional bust, and (2) Artistic to give drawing in two views, facial and profile, to show cephalic and facial details. Both methods try to achieve an individual likeness which will lead to identification of the suspect.

SKULL: When previous X-ray films of the skull are available for comparison with postmortem films, measurements of the skull and a detailed comparison of the frontal, sphenoidal and maxillary sinuses, sella turcica and mastoid area are useful for identification in adults.

VEINS: The arrangement of the veins of the back of the hands, is said to be individual.

STOMACH PICTURES: Barium meal X-ray of the stomach is said to be individual.

X-RAYS: (1) They are useful in identification by determining the sex, age, height of the person, superimposition and by detecting old fractures, diseases or congenital bony changes. (2) In investigation of cases of battered baby syndrome,

burns, drowning and decomposition. (3) To detect air embolism, and pneumothorax. (4) To locate missiles in the body, direction of firing, depth of the wound and type of firearm. (5) To detect parts of the mechanism of bombs and explosive devices those are embedded in the tissues. (6) To locate foreign bodies, bullet emboli, etc. (7) To detect fractures. (8) To detect pregnancy, foetal death or abnormality, hydatidiform mole, etc. (9) Electrocution. (10) Lead poisoning. (11) Drug smuggling through sealed packets in body cavities, gut and genitalia. (12) Road traffic accidents to understand and record pattern of skeletal injuries. (13) Medical negligence cases where embolism (air, fluid, amniotic), wrong placement of endotracheal tube, leaving of surgical instruments is suspected.

NAILS: The character of the nail-bed is highly individual, and the parallel striations on the surface of the fingernail are significantly different for each individual. The striation pattern remains unchanged during the life of the person.

DEFORMITIES: They may be congenital or acquired. Congenital deformities, such as harelip, cleft palate, talipes, polydactylism, webfingers or toes, undescended testicles, etc., are frequently treated surgically, and are losing some of their past importance. Old amputations, spinal defects, old fractures and deformities of the bones and nails, either from injuries or disease and surgical prostheses, such as implanted artificial heart valves, plates in the skull, etc., should be described.

DACTYLOGRAPHY

Introduction: (Fingerprint system; dermatoglyphics; Galton system). **Dermatoglyphics is the study of ridge patterns in the skin.** This system was first used in India in 1858 by William Herschel in Bengal. Sir Francis Galton systematised this method in 1892.

Principle: Fingerprint Bureau was first established in Kolkata. **Fingerprints are impressions of patterns formed by the papillary or epidermal ridges of the fingertips.** The ridge pattern of fingers appears between 12 and 16 weeks of intrauterine life and the formation is completed by 24 weeks. **At birth, a fine pattern of ridges is seen on the skin of the bulbs of the fingers and thumbs, parts of the palms and the soles of the feet.**

Classification: (1) Loops (about 60 to 70%). (2) Whorls (about 25 to 35%): (3) Arches (about 6 to 7%), and (4) Composite form (about one to 2%) **(Fig. 4-29)**.

Identifications: The final identification of any fingerprint is not made by comparison of patterns. It is made by comparison of many details of characteristics which occur throughout the ridge areas, and by the sequence in which these characteristics occur. The characteristics may take the form of ridge endings, bifurcations, lake formations, or island formations. **In practice 10 to 12 points of fine comparison are accepted as proof of identity.** The **patterns are not inherited and paternity cannot be proved through fingerprint patterns. The pattern is different even in identical twins.** The fingerprint **patterns are distinctive and permanent in individuals.** The details of these can be accurately teleprinted. **The fingerprint system is the only guide to identity which is unfailing in practice.** Palm and footprints also provide similar material.

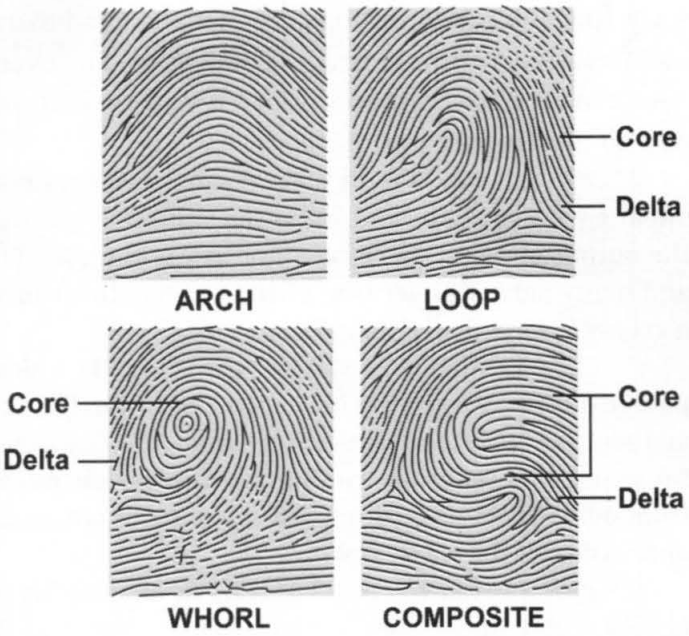


Fig. (4-29). Four main fingerprint patterns.

POROSCOPY: This is the further study of fingerprints described by Locard. **The ridges on fingers and hands are studded with microscopic pores, formed by mouths of ducts of subepidermal sweat glands.** Each millimetre of a ridge contains 9 to 18 pores. These pores number in thousand per square centimetre. These **pores are permanent and do not change during life** and vary in size, shape, width, starting and stopping on occasion, branching at points, position, extent and number over a given length of ridge in each individual. This **method of examining pores is called poroscopy, and is useful when only fragments of fingerprints are available**.

The Individuality of Fingerprints: The fingerprints are capable of endless variation, so that it has been speculated that there is one chance in

sixty four thousand millions, of two persons having identical fingerprints. Quetelet's law states that every nature made object presents infinite variation in forms and no patterns are ever alike.

MODE OF PRODUCTION: A constant stream of sweat covers the skin and when the subject is excited, the output of sweat increases. The sweat contains fat, and if any part of finger is applied to a smooth surface, a greasy impression of its pattern is made on it.

TECHNIQUES OF FINGERPRINTING: (1) A plain print is taken by applying ink to the tips of the fingers, and placing the fingers directly on paper. (2) The rolled fingerprint is taken by rolling the fingers on paper from outward to inward in such a way as to obtain an impression of the whole tip.

TYPES: (1) The LATENT PRINT, is an invisible or barely visible impression left on a smooth surface. (2) VISIBLE PRINTS, formed by fingers stained with blood or ink or other medium. (3) The PLASTIC PRINT, is an impression made on a soft surface such as soap, cheese, etc. Fingerprints may be taken from almost any surface of contact, including certain fabrics and human skin.

DEVELOPMENT OF LATENT PRINTS: A latent print may be developed by dusting the area with coloured powders, such as grey powder (chalk and mercury), and its pattern is recorded by photography. It can also be examined by oblique lighting. Fingerprints on paper can be developed by exposing it to the vapours of iodine or osmium tetroxide.

LIFTING: The fingerprint is dusted and a special clear adhesive tape is pasted over the treated print. When the tape is removed, the print comes away with it, which can be photographed.

ASSAILANTS FINGERPRINTS: The fingerprint of an assailant will remain on the victim only after death. During pre-death struggle, both the assailant and victim will sweat, and one exudate will cancel out the other.

FINGERPRINTS IN DECOMPOSED BODIES: Ridges are present both in dermis and epidermis. In advanced putrefaction and in cases of drowning, the skin is frequently found loose like glove, which should be removed, preserved in formalin and used for impressions. Prints can be obtained from the dermis after the epidermis is lost. Histological sections up to a depth of 0.6 mm from the surface of the skin give satisfactory fingerprints. In dead bodies, the palmar skin of terminal phalanx of each finger should be removed separately from both hands, and after labelling, placed in separate containers containing 10% formalin, and sent to the fingerprint bureau, if it is not possible to take the prints.

PERSISTENCE OF IMPRESSION AT SCENE OF CRIME: Impression may persist for years if undisturbed by cleaning. Even outside the house, they may persist for weeks. On glazed paper, they persist for more than three years.

MUTILATION OF FINGERPRINTS: Criminals sometimes attempt to mutilate the pattern by self-inflicted wounds or burns, application of corrosives or erosion against a hard surface, but they are not destroyed unless the true skin is completely destroyed. They produce additional characteristics. It leaves some part of the skin undamaged, unless skin grafts are made. Permanent impairment of the fingerprint pattern occurs in leprosy, electric injury and after exposure to radiation.

Computerisation: Fingerprint reader (FINDER) is a computerised automatic fingerprint reading system which can record each fingerprint data in half second. Prints of eight fingers are recorded excluding little fingers. **The light reflected from a fingerprint can be measured and converted to digital data which is classified, codified, and stored in the computer.**

Medicolegal Importance: (1) The recognition of impressions left at a scene of crime, e.g., on weapons, furniture, doors, utensils, clothes, etc. establish the identity of the criminal. (2) The identification of suicides, deserters, persons suffering from loss of memory, or those dead or unconscious after being involved in an accident and of decomposing bodies. (3) Identification in case of accidental exchange of newborn infants. (4) The prevention of impersonation. (5) To maintain identity records. (6) Cheques, bank notes and other legal documents can bear a fingerprint.

In criminals, impressions of all the ten fingers are taken, but for civil purposes the left thumb impression only is taken.

FOOTPRINTS: The skin patterns of toes and heels are as distinctive and permanent as those of the fingers. Footprints of newborn infants are used in some maternity hospitals to prevent exchange or substitution of infants. A fresh footprint of suspected person is taken and compared with the original. Any peculiarities in the foot, such as a flat foot, supernumerary toes, scars or callosities, are likely to be found in the footprint. In case of boot mark, the pattern and arrangement of nails or holes in the sole may be useful. A footprint produced by walking is usually larger than one produced by standing. The imprint on soft and loose material like sand is smaller than the foot, and the imprint produced on mud or clay is larger.

PALATOPRINTS (Podogram): In the anterior part of the palate, the structural details, such as the rugae, are individual specific and permanent. The palatoprints can be used in the same way as fingerprints.

LIP PRINTS (Cheiloscopy): Lip prints are divided into eight patterns which are specific to the individual. The fissures and grooves on the lips are claimed to be characteristic of the individual. 24 characteristic details have been identified. Identification is established if 7 to 9 characteristics tally.

EAR PRINTS: The shapes of ear lobes and the tips of ears are of various types. The transparencies of ear print at the scene and of the ear of the suspected person are overlaid and compared.

NOSE PRINTS: The lines on the nose and the shape of the tip are helpful in identification.

RETINA IDENTIFICATION: Each person's retina has an unique pattern which is not changed from birth until death. The pattern is different even in identical twins. As such, it appears to be the most precise and reliable biometric. A retinal scan is performed by casting an undetectable ray of low-energy infrared light into a person's eye as he looks through the scanner's eyepiece, focusing on a single point for a duration of 15 seconds, which outlines a circular path on the retinal. Retinal blood vessels absorb light more readily than the surrounding tissue, but the amount of reflection fluctuates. The results of the scan are converted to computer code and stored in a data base. Using suitable cameras and infrared illumination, image of cornea is copied and converted into digital templates. The retinal template is one of the smallest of any biometric technology. An enrolled person can be identified with a retinal scan process in seconds. They are about 70 times more accurate than iris scan. They are nearly impossible to fake. After death, retina decays quickly. Retinal patterns may be altered in cataracts, glaucoma, retinal degeneration and diabetes.

IRIS SCAN: The iris is a muscle within the eye that regulates the size of the pupil controlling the amount of light that enters the eye. It is the coloured portion of the eye and the colouring is based on the amount of melatonin pigment within the muscle. Although the colouration and structure of iris are genetically linked, an individual's irises are unique and structurally distinct, which allows for them to be used for recognition purposes. The iris is located using landmark features. The distinct shape of iris allows for imaging feature isolation and extraction.

SCARS

A scar is a fibrous tissue covered by epithelium, without hair follicles, sweat glands or pigment, and results from the healing of a wound. **Injury to the dermis produces a scar**, while superficial injuries involving only the epidermis do not produce a scar. Scars are permanent.

Examination: The description should include their number, site, size and shape, fixed or free, smoothness or irregularity of the surface, colour and the presence or absence of glistening and tenderness. The condition of the ends, whether tapering or not, and the probable direction of original wound should be determined. The application of heat, filtered ultraviolet light or surface friction, makes faint scars readily visible. Old scars may become unrecognisable. A magnifying glass is very useful.

CHARACTERS: Scars may indicate the type of injury which produced them. (1) Incised wounds produce linear scars. If healing is secondary, the scar is wider and thicker in the centre than in the periphery. (2) Scars from lacerated wounds, and from wounds which have suppurated are firmer, irregular, more prominent, and are attached to the deeper tissues. (3) Stab wounds due to a knife-blade produce oval, elliptical, triangular, or irregular scars which are depressed, but may be elevated due to keloid formation. (4) A bullet wound causes a circular depressed scar. (5) Scars from scalds are spotted in appearance, tend to be continuous, often run downwards and show evidence of splashing about the main injury. (6) Scars due to corrosive acids, burns or radiation, cause irregular scars, and keloid may develop in the scar tissue especially in Negroes. (7) Vaccination scars are circular or oval, flat or slightly depressed. (8) Many skin diseases, smallpox, syphilis, etc., cause multiple scars on the skin.

Growth: Scars produced in childhood grow in size with the natural development of the individual, especially if situated on the chest or limbs.

Age of Scars: Firm union occurs in from 5 to 6 days, producing a reddish or bluish "angry" scar. By the end of 14 days the scar becomes pale; it is still soft and sensitive; there is no further change up to the end of the second month. In about 2 to 6 months, the scar becomes white and glistening, tough and may be wrinkled. After this there is no further change.

ERASURE: The scar can be erased by excision and skin grafting or suture of the edges of the excised area. This results in a scar which is less obvious.

Medicolegal Importance: (1) They form important marks for identification of a person. (2) The shape of the scar may indicate the nature of weapon or agent that caused the injury. (3) The age of the scar is important in a criminal offence. If the age of a wound corresponds with the date of the attack, it may have value as circumstantial evidence. (4) Linea albicantes may indicate previous pregnancy. (5) Disfiguration of the face or head is a grievous injury. (6) Scar causing contracture at or around a joint restricting movement of functions of the joint becomes grievous hurt.

Mongolian spot (congenital marks) appears as dark blue or violet, single or multiple macules in the lumbosacral region in some young children of Asiatic and African origin.

Moles are usually round, brown or black, raised or flat, with or without hair.

TATTOO MARKS

Mode of production: Tattoo marks are designs imprinted in the skin by multiple small puncture wounds with needles or an electric vibrator dipped in colouring matter. The **dyes commonly used** are: Indian ink, carbon (black); cinnabar or vermilion (mercuric sulphide) (red); chromic acid (green); indigo, cobalt, Prussian blue (ferric ferrocyanide), ultramarine (blue). Techniques and dyes vary from country to country. **The most permanent pictures are made when the dye penetrates the dermis.** Most of the marks are found on the arms, forearms and chest, but may be present on any part of the body.

Natural disappearance: If the pigment has been deposited below the epidermis, it will very slowly become fainter, and certain pigments, such as vermilion, cinnabar and ultramarine, may disappear after ten years. Tattoos on the hands disappear early due to constant friction. A faded tattoo mark becomes visible by the use of ultraviolet lamp or rubbing the part and examining with magnifying lens. Infrared photography makes old tattoos readily visible.

The marks are recognised even in decomposed bodies, when the epidermis is removed. Lymph nodes near a tattoo mark show a deposit of pigment. The colour, design, size and situation of tattoo marks should be noted. Drawing or photography is more useful.

COMPLICATIONS: Septic inflammation, erysipelas, abscess, gangrene, and even syphilis, leprosy, AIDS, and tuberculosis may occur.

ERASURE: (1) SURGICAL METHODS: (a) Complete excision and skin grafting, (b) production of burn by means of red hot iron, (c) scarification, and (d) carbon dioxide snow. (2) ELECTROLYSIS. (3) CAUSTIC SUBSTANCES remove pigment by producing inflammatory reaction and a superficial scar.(4) LASER BEAM. (5) Confluent smallpox and sometimes chronic eczema in children can obliterate tattoo marks.

Medicolegal Importance: Tattooing is useful in (1) Identity: Initials and dates, regimental or nautical details, identity numerals, one's own name, etc., provide more scientific basis for identification. (2) Religion. (3) God of worship. (4) Indicate culture or lifestyle. (5) The distribution of tattooing and the nature of designs and drawings may be characteristic of a particular country or region. (6) The presence of indecent figures points to perversion in the individual. (7) They reflect travel, history, war, occupation, sex interest, etc. (8) Gang members may wear a tattoo of allegiance and symbolism to indicate status or other aspects relevant to their particular group. (9) Illicit drug users may have tattoos that identify them as belonging to a particular group or to obscure injection sites. (10) Homosexuals may tattoo on the back of hand between base of thumb and forefinger.

WOUNDS: Sometimes, the presence of wounds on the body may assist in connecting a suspected criminal with a given crime, e.g., a piece of skin adherent to a window glass may correspond with the wound on the thief, or rupture of fraenum of penis may be present in a person accused of rape. Particles of stone, sand, dust, etc., may be recovered from wounds and identified.

DISEASE: The finding of disease, e.g., gall stones, renal stones, calcified leiomyomata, silicosis, asbestosis and congenital anomalies, like horseshoe kidney, the absence of certain organs, such as the appendix, uterus or gall bladder, etc., can be compared to such abnormality known to be present in a missing person.

STAINS: Stains found on body or clothing of the accused and the victim may be the same, and be derived from the walls, doors, furniture, etc., at the scene of crime.

OCCUPATION MARKS: (1) RECENT AND TEMPORARY: They include paint spots in painters, grease on engineers and mechanics, flour on bakers and millers, dyes on dye workers, etc. (2) PERMANENT: A callosity is seen on the outer side of the distal phalanx of the middle finger of the right hand of a clerk. Blacksmiths have scars on the back of the hands caused by burns from hot fragments. Opticians have small

cuts on the tips of index finger and thumb. Cuts, scars, callosities and hyperkeratosis of the hands are seen on the right hands of butchers. Horny and rough hands are seen in manual labourers. Tailors have marks of needle punctures on their left index finger. *Dooly* bearers usually have horny callous marks on their shoulders.

COMPLEXION AND FEATURES: The complexion may be fair, wheat-coloured, dark, brown, pale-brown or pale-yellow. Details of the features regarding eyes, nose, ears, lips, chin and teeth should be noted. The features may change considerably from disease or even from worries of a long duration. Few persons can cleverly alter their features by changing the expression of their face. Expression is altered after death.

HAIR

Trichology is the study of hair. Hair grows at the rate of 0.4 mm/day and nails at 0.1 mm/day.

(1) HAIR OR SOME OTHER FIBRE? MORPHOLOGY: Hair consists of bulb or root and a shaft. (1) Cuticle: This is the outer layer and consists of thin, non-pigmented scales. (2) Cortex: This is the middle layer and consists of longitudinally arranged, elongated cells without nuclei. Within these cells are fibrils on which there may be granules of pigment. (3) Medulla: This is the inner layer composed of keratinized remains of cells (Fig. 4-30).

(2) HUMAN OR ANIMAL HAIR? (Table 4-8 and Fig. 4-31). Colour changes along the hair shaft called "banding" is seen in some animals.

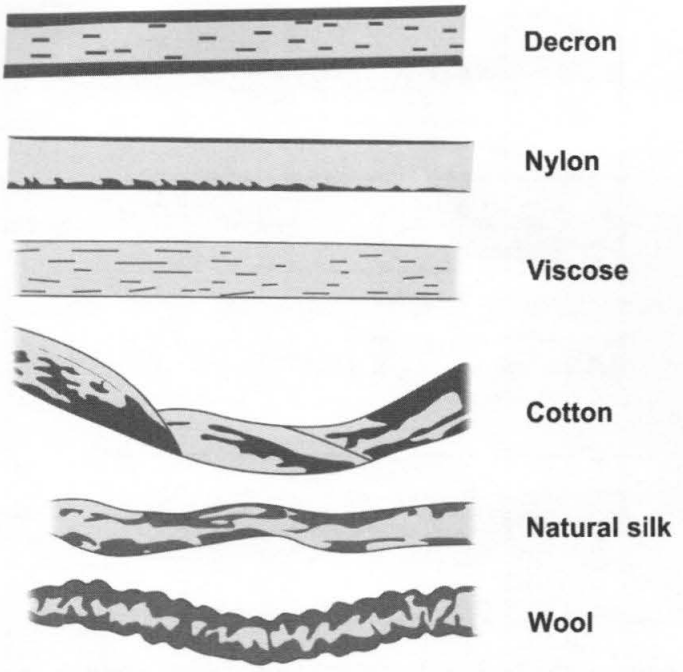


Fig. (4-30). Microscopic appearance of fibre.

(3) FROM WHAT PART OF THE BODY DERIVED?

Hair from the head is usually long and soft, and taper gradually from the root to the tip. The beard and moustache hair are usually thicker. Hair of the eyebrows, eyelashes and nostrils is stiff, thick and taper to a point. The hair on the chest, axillae and pubic region is short, stout and curly. Those from the axillae and pubic region also show split ends. The hair on the other

Table (4-8). Difference between human and animal hair

Trait	Human hair	Animal hair
General:	Fine and thin.	Coarse and thick.
Cuticle:	Cuticular scales are short, broad, thin and irregularly annular.	Cuticular scales are very large and have step-like or wavy projections.
Cortex:	Thick, well striated, and four to ten times as broad as medulla.	Thin, rarely more than twice as broad as medulla.
Medulla:	Varies considerably, usually narrow; continuous, fragmented or entirely absent.	Continuous and wider.
Pigment:	Evenly distributed.	Mostly present near medulla.
Precipitin test:	Specific for human.	Specific for animal.
Medullary index: $\left(\frac{\text{Diameter of medulla}}{\text{Diameter of shaft}}\right)$	Less than 0.3.	More than 0.5.

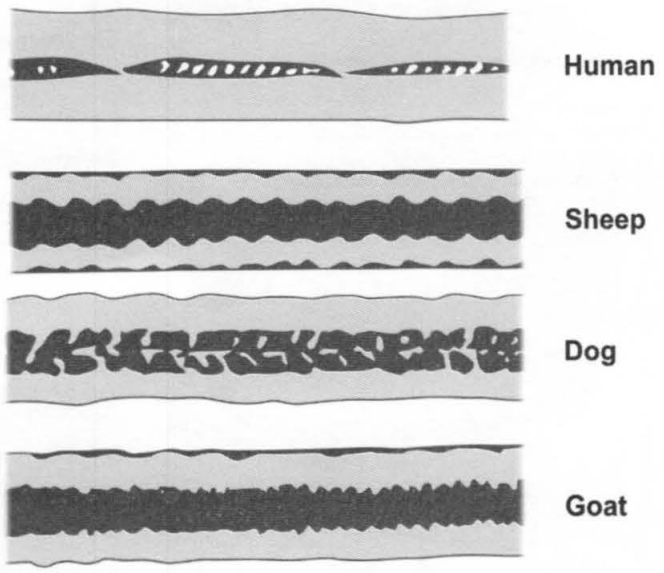


Fig. (4-31). Various hair.

parts of the body is fine, short and flexible and does not show pigment cells in the cortex.

(4) Sex: Sexing of human hair is difficult, except that of the beard and moustache. Male hair is usually thicker, coarser and darker. In human head hair, **Barr bodies** are found in hair follicles in a proportion of 29 ± 5 per cent in females and 6 ± 2 per cent in males.

(5) Age: Roots of hair from children will dissolve rapidly in a solution of caustic potash, but in older people roots will resist the treatment. The body hair of the human foetus and the newly born child is fine, soft, non-pigmented (colourless) and non-medullated. This **lanugo hair** is replaced, by hair which is coarser, pigmented, medullated, and has a more complex scale pattern. At puberty, axillary and pubic hair grows which is at first fine, soft and curly, and later becomes coarse and pigmented. Adult hairs have maximum pigmentation. Loss of scalp hair in men starts from the third decade. In women, there is often loss of axillary hair and an increase of facial hair at about the menopause. Grey hair usually appears after forty years.

(6) Has the hair been altered by dyeing, bleaching or disease? Bleached hair is brittle, dry and straw-yellow. If the hair is coloured, the colour will not be uniform, the roots being of different colour and the hair rough, brittle and lustreless. The scalp will also be coloured. The colour of head hair will be different from the colour of hair on other parts of the body. Scalp hair grows at the rate of two to three mm. a week, average being two-and-half mm; beard hair has a slightly faster and other hair a slightly slower growth rate. Some hair can be examined chemically to find out any metal contained in the paint.

Blood Groups: ABO groups can be determined in a single hair from any part of the body by modified absorption-elution technique.

IDENTIFICATION: Hair cannot provide a permanent record for identification, because the distribution and concentration of trace elements along the shaft of a hair varies as the hair grows.

(7) IS THE HAIR IDENTICAL WITH THE HAIR OF THE VICTIM OR THE SUSPECT? By careful comparison one can say that the hair could have come from a particular person. Debris, grease, etc., adherent to the hair is very important.

(8) DID IT FALL NATURALLY OR WAS IT FORCIBLY REMOVED? The base must be examined to see whether the root is present. If the hair has fallen naturally the root will be shapeless and atrophied and the root sheath absent. If the hair is forcibly pulled out, the hair bulb will be larger, irregular and swollen and the sheath will be ruptured.

(9) WHAT IS THE CAUSE OF THE INJURY? If the hair has not been cut, the tip is pointed and non-medullated. Repeated injury to the tip damages the cuticle due to which the exposed and unprotected cortex splits and becomes ragged. The hair of axilla, pubis and frequently brushed hair has ragged ends. Blunt injury and crushing with heavy blunt scissors, crushes the shaft with flattening and splitting. A sharp weapon produces a clean uniform cut surface. Recently cut hair shows a sharply cut edge with a projecting cuticle. After a week the end becomes square, smooth and later rounded but blunt. After three to four months, the end becomes elongated, but the medulla is absent. Hair may get singed due to burns or firearm injury. Singed hair is swollen, black, fragile, twisted or curled and has a peculiar odour; carbon may be found deposited on it. The tip is swollen like a bulb.

Medicolegal Importance: (1) Hair is important in crime investigation, for it remains identifiable on the clothes, body, and the alleged weapons in crimes committed some time back. It often provides the only connection between a weapon, or even the accused and the victim of an assault. (a) Motor vehicles responsible for injuries may be identified by the detection of hair on the vehicle. (b) In rape and sodomy, the pubic hair of the accused may be found on the victim or vice versa. In bestiality, animal hair may be found on the body or underclothing of the accused, or his pubic hair may be found about the genitals of the animal. (c) Stains on the hair may indicate the nature of the assault, e.g., mud stains in struggle, seminal stains in sexual offences, salivary stains in

asphyxial deaths, blood stains in injury, etc. Stains may be got from the walls, doors, furniture, etc., and may indicate the scene of crime. (2) Nature of weapon can be made out from the injuries to the hair, and hair bulb. (3) Hair is useful in identification especially when there has been some known peculiarity of the hair, dyeing, bleaching, or artificial waving. (4) Age of a person may be determined from the growth of hair on different parts of body. (5) Sex and blood group may be determined from their distribution on body, texture, and from Barr bodies. (6) Singeing of the hair indicates burns or a close range firearm injury. (7) It is helpful in differentiating scalds from burns. (8) In chronic poisoning with heavy metals, e.g., arsenic, the poison can be detected in the hair. (9) The time of death can sometimes be determined from the length of the hair on the face.

FORENSIC ODONTOLOGY

Forensic odontology deals with the science of dentistry, to aid in the administration of justice. Dental identification depends mainly upon comparison between records of the missing persons and the findings in the bodies in relation to: (1) Restorative work. (2) Unusual features. (3) Comparison of antemortem with postmortem X-rays.

IDENTIFICATION OF AN INDIVIDUAL: **The following particulars should be noted: (1) The number, spacing and situation of the teeth present, with special note of (a) unerupted and deciduous teeth, (b) permanent teeth, (c) decayed teeth, (d) undersized or oversized teeth. (2) The number and situation of absent teeth. (3) Extraction: evidence of old or recent, healed or unhealed. (4) The general condition of teeth: (a) erosion, (b) cleanliness, (c) conservation, filling and cavities, (d) colour, (e) periodontosis. (5) Peculiarities of arrangement: (a) prominence, (b) crowded or ectopic teeth, (c) overlapping, (d) malposition, (e) deformities, (f) rotation. (6) Supernumerary teeth. (7) Denture: full, partial, upper or lower, type, shape. Restorative materials used. (8) Mesiodistal width of the teeth. (9) Any recognisable peculiarity of jaws, e.g., prognathism (prominence of the lower jaw). (10) Old injury or disease. (11) Special features, incisal edges, fractures, ridges, caries, etc. (12) Restoration and prostheses. (13) Root canal therapy on X-ray examination. (14) Oral pathology.**

Medicolegal Importance: **Dental identification is the most sophisticated method of comparative identification, except dactylography, if there are some features to compare and some record of those features in a missing person.** It is not of much help in developing countries as dentists often do not keep records. (1) Definite identification of a body from dental data can be made if accurate and dated dental records are available. Identification is possible even in burnt bodies (**Table 4-9**). (2) Dentures, partial or complete, are useful in identification, especially if they have the patient's name or code number included in them. (3) Criminals can be identified through bite-marks left either in human tissues or in food stuffs. (4) Teeth are useful in estimating the age of an individual. (5) Sex can be determined from cells of pulp cavity. (6) Loss of tooth due to assault is grievous hurt. (7) Heavy metal poisoning can be detected. (8) Colour change occurs in sulphuric and nitric acid poisoning.

Table (4-9). Advantages and disadvantages of comparisons of teeth and fingerprints.

Trait	Teeth	Finger prints
(1) Burns:	Fire resistant.	Destroyed by fire.
(2) Putrefaction:	No changes.	Subject to putrefaction.
(3) Changes:	Compatible inconsistencies.	Unchanged.
(4) Proof:	No acknowledged criteria of proof.	Well established criteria.
(5) Records:	Useless without records.	Possible use of possessions.

ANTEMORTEM TOOTH LOSS OR EXTRACTION: **The bony rim of the alveolus is sharp and feathered. A blood clot forms within the alveolar cavity and in one to two days there is early organisation. In about a week, socket is filled with organised clot. In two to three weeks soft tissues are healed and the socket is partially filled with new bone. Reparative bone resorption of the alveolar rim results in a smooth rounded rim of socket. In six months, the socket is filled with new bone, but the location of the root outline is visible. In one year the whole socket is filled with a new bone, and there is depression of the bone outline. If the entire tooth was knocked out, irregular edges of remaining bone, splintering of buccal or lingual plates, areas of compressed bone, or fracture of roots or crowns of adjacent teeth are seen. It is difficult to dislodge a healthy tooth without fracturing or loosening neighbouring teeth.**

Charting of Teeth: There are more than 150 different methods of identifying, numbering, and charting of teeth. The most widely used systems are:

(1) Universal system: Teeth are numbered 1 to 16 from upper right to upper left, and 17 to 32 from lower left to lower right. This follows the plan advocated by the American and International Societies of Forensic Odontology.

Right $\frac{1\ 2\ 3\ 4\ 5\ 6\ 7\ 8 \mid 9\ 10\ 11\ 12\ 13\ 14\ 15\ 16}{32\ 31\ 30\ 29\ 28\ 27\ 26\ 25 \mid 24\ 23\ 22\ 21\ 20\ 19\ 18\ 17}$ Left

(2) Palmer's notation:

Right $\frac{8\ 7\ 6\ 5\ 4\ 3\ 2\ 1 \mid 1\ 2\ 3\ 4\ 5\ 6\ 7\ 8}{8\ 7\ 6\ 5\ 4\ 3\ 2\ 1 \mid 1\ 2\ 3\ 4\ 5\ 6\ 7\ 8}$ Left

(3) Haderup System. It is similar to Palmer's notation except that it uses a plus sign (+) to designate upper teeth, and a minus sign (–) for the lower.

(4) FDI (Federation Dentaire Internationale) two-digit system: It gives a number for the quadrant side and that number is placed before the tooth number. Thus the lower right canine will be number 43 in this system.

Right $\frac{1 \mid 2}{4 \mid 3}$ Left

Modified FDI system:

Right $\frac{1 \mid 2}{3 \mid 4}$ Left

Radiography: X-rays accurately reveal root shape, shape of pulpal canal, shape of fillings, abnormalities, bone trabeculation patterns, caries, tooth formation, and fractures. They are widely used in antemortem and postmortem comparisons **(Table 4-9)**.

BITE MARKS (Plate 1-3): They are useful in identification, because the alignment of teeth is peculiar to the individual. Bite marks may be found in materials left at the place of crime, e.g., food stuffs, such as cheese, bread, butter, fruit, or in humans involved in assaults, when either the victim or the accused may show the marks. Bite marks can be found anywhere on the body, but are usually seen on the breasts of girls as love bites. Human bites seldom cause tearing of the skin, but frequently produce semicircular or crescentic patterned abrasions with extensive haemorrhages beneath the skin. The mark consists of a central area of bruising with a surrounding area, which may show separate bruises and abrasions. Bites are always contaminated with saliva, and as such, blood groups can be determined. Teeth bites can produce abrasion, contusion and laceration.

ANIMAL BITES: Rodents gnaw away (bite persistently) tissue over fairly limited areas. They produce shallow craters and leave long grooves. The bites by dogs which attack suddenly are usually clear-cut. Teeth impressions are usually deep and small in area. Cat bites are usually very small and round.

CLOTHES AND ORNAMENTS: The clothing may indicate the social status to a certain extent from the texture and value. Any variety of uniform is very valuable for identification. Clothing may also indicate the occupation. The examination of clothing and personal effects is helpful in the identification of victims in mass disasters, such as fires, explosions and aircraft crashes. A detailed description of the size, colour, condition and type of each garment and a record of laundry marks, name tags and labels of tailors, should be given. The clothing may contain keys, letters, bank books, visiting cards, licenses or other documents, which may give a clue to the name and address of the individual. Other personal effects like watches, rings, keys, belt buckle, etc., may be engraved with initials, names or dates. Eye glasses may also be helpful. Bullet holes, tears, cuts or tyre marks found on clothing may give information regarding the cause and manner of death. The design of the ornaments varies from region to region. A criminal may interchange his identity with that of another person by clothing and personal effects.

HANDWRITING: Handwriting is characteristic of the individual, especially if it is written rapidly, but it may be forged. Mental and nervous disease and rheumatism alter the character.

SPEECH AND VOICE: Certain peculiarities of speech, e.g., stammering, stuttering, lisping and nasal twang become more evident when the individual is talking excitedly. Speech is also affected by nervous disease. To recognise a person from the voice is risky. It is possible for a person to alter his voice at will. Tape recording is useful. No two voices are really alike.

GAIT: Identification based on recollection of physical characteristics of person in question by friends and relatives is unreliable. The gait may be altered by an accident or by design.

TRICKS OF MANNER AND HABIT: They are frequently hereditary, e.g., left-handedness. Jerky movement of shoulders or muscles of face is an individual characteristic.

MEMORY AND EDUCATION: They are sometimes useful especially in cases of imposture.

CHAPTER

5 Medicolegal Autopsy

Autopsy or necropsy means postmortem examination of a body. In every case the autopsy must be complete, all the body cavities should be opened, and every organ must be examined.

Objects: (1) To find out the **cause of death**, whether natural or unnatural. This is done by detecting, describing and recording any external and internal injuries, abnormalities and diseases. (2) To find out the **time since death**. (3) To find out the **manner of death**, whether accidental, suicidal or homicidal. (4) To establish **identity** when not known. (5) To collect **physical (trace) evidence** in order to identify the object causing death and to identify the criminal. (6) In newborn infants to determine the question of **live birth and viability**.

Rules for Medicolegal Autopsies: (1) The autopsy should be conducted in a mortuary and never in a private room. However, it may become necessary to do an autopsy at the site, when the body is in an advanced state of putrefaction and its transportation will be difficult. (2) It should be conducted only when there is an official order from the police or Magistrate. (3) It should be conducted as soon as possible after receiving requisition, without undue delay. (4) The medical officer should first read the inquest report carefully, and find out the apparent cause of death and obtain all the available details of the case sheet, accident register, etc., so that attention may be directed to the significant points. (5) The examination should be conducted in daylight as far as possible, because colour changes, such as jaundice, changes in bruises, changes in postmortem staining, etc., cannot be appreciated in artificial light. **There is no law which prevents autopsy being conducted during night**. (6) The body must be identified by the police constable who accompanies it. (7) No unauthorised person should be present at the autopsy. The investigating police officer may be present. (8) During autopsy, details of examination should be noted verbatim by an assistant, or use a voice-activated recorder, and sketches made of all the important injuries. (9) Nothing should be erased, and all alterations should be initialled in the report. (10) In every case the autopsy must be complete, and every organ must be examined. (11) Even if the body is decomposed, autopsy should be performed as certain important lesions may still be found. (12) After completion of autopsy, the body is handed over to the police constable. (13) **P.M. report should not be issued to the party.**

The approach of the forensic pathologist to the investigation of death is different from that of the hospital pathologist. THE HOSPITAL PATHOLOGIST has easy access to relevant information about the history, physical condition and course of the disease leading to death. His main aim is to find morphologic changes explaining signs or symptoms of the disease. FORENSIC PATHOLOGIST has to determine time of death and age of injuries. In medicolegal autopsies, often the clinical history is absent, sketchy or doubtful. In some cases, identity may not be known. If there are any inconsistencies between the apparent death scene and his actual findings, he has to visit the scene of crime. He has to carry out careful external examination including clothing, in the determination of the pattern of injuries and their relationship to the object or weapon causing them. He has also to determine the manner and mechanism of death. The autopsy should be carried out by the doctor, and not left to the mortuary attendant.

AUTOPSY REPORT: It consists of: (1) THE PREAMBLE: This should mention the authority ordering the examination, time of arrival of the body at the mortuary, the date and place of examination, the name, age and sex of the deceased. (2) THE BODY OF THE REPORT: This consists of a complete description of the external and internal examination of the body. (3) CONCLUSIONS: The conclusion as to the cause of death must be given, based on the postmortem findings. This is followed by the signature and qualifications of the medical officer.

EXTERNAL EXAMINATION: The following should be noted: (1) The clothing should be listed, examined and described with regard to type of garment, its colour and consistence, tears, loss of buttons or disarrangement indicating a struggle, as each item is removed from the body. Clothing should be removed

gently taking care to avoid contamination, or less of any trace evidence, such as hair, fibres, paint fragments, glass, sand, vegetable matter, etc., which should be collected, mentioning the site, sealed and handed over to the police. The clothes should be removed carefully without any tearing to avoid confusion of signs of struggle. If they cannot be removed intact, they should be cut in an area away from any bullet hole or objects, along a seam in the garment. Clothing removed from the victim should not be thrown on the ground or floor or otherwise discarded or destroyed. They should be handled as little as possible and without any deliberate shaking or dusting. Cuts, holes or blackening from firearm discharges should be noted and compared with the injuries on the body. No object should be put through defects in clothing or wounds. Blood stains, seminal stains, grease stains, etc., should be described. Stains due to poison, vomit etc. should be kept for analysis. Wet clothing should be hung up to dry, but should not be heat dried. Packing of wet clothes may promote growth of fungus. Stained and unstained areas of clothes should not be allowed to come in contact to avoid additional soiling, and as such clothes should not be folded while stains are wet. The clothes should be placed into clean plastic bags or other suitable clean containers. Separate bags or containers should be used for each article. (2) Height and weight of the body, body build, development and nourishment. (3) General description: This includes sex, age, colour, deformities, hair, scars, tattoo marks, moles, pupils, etc. (4) General condition of the skin (rash, petechiae, colour, looseness, turgor), disease, asymmetry of any part of the body or muscle wasting. (5) Note the presence of stains on the skin from blood, mud, vomit, faeces, corrosive or other poisons or gunpowder. (6) The presence of signs of disease, e.g., oedema of legs, dropsy, surgical emphysema about the chest, etc., is to be noted. (7) The time since death should be noted from rectal temperature, rigor mortis, postmortem hypostasis, putrefaction, etc. (8) The face should be examined for cyanosis, petechial haemorrhages, pallor, etc. (9) The eyes should be examined for the condition of the eyelids, conjunctivae, state and colour of pupils, artificial eyes, petechiae, and for extravasation of blood. (10) The neck must be examined for bruises, fingernail abrasions, ligature marks or other abnormalities. (11) The natural orifices, i.e., mouth, nostrils, ears, vagina, etc., should be examined for injuries, foreign matter, blood, etc. The state of the lips, gums and teeth, marks of corrosion, and injuries to inside of lips, gums and cheeks should be noted. The state of the tongue, position with relation to the teeth, and the presence or absence of bruising or bitemarks should be noted. The presence of froth about the mouth and nostrils and smell of alcohol, phenol, etc., should be noted. (12) Needle puncture marks in the arms, buttocks, etc. should be looked for. (13) The limbs and other parts should be examined for fractures and dislocations by suitable movements and by palpation and confirmed by dissection. (14) The report should include all of the surgical procedures, applied dressings, and other diagnostic and therapeutic measures found on external examination. (15) A list should be made of all articles removed from the body, e.g., clothes, jewellery, bullets, etc. They should be labelled, mentioned in the report and handed over to the police constable in a sealed cover and a receipt is taken. **(16) External wounds should be systematically examined taking up each part of the body in turn. The description of wounds should include nature, site, length, breadth, depth, direction, margins, base, and extremities. The condition of their edges, coagulated blood and evidence of bleeding into nearby tissues noted. Determine whether they were caused before or after death and their time of infliction. Collect foreign materials, e.g., hair, grass, fibres, etc., that may be in the wound. Sketches and photographs are useful. Excluding stab and firearm wounds, all the injuries should be divided into two broad areas: external and internal.**

SKIN INCISIONS: Primary incisions are of three types. (1) The "I" shaped incision, extending from the chin straight down to the symphysis pubis passing either to the left or right of the umbilicus. (2) "Y" shaped incision begins at a point close to the acromial process. It extends down below the breast and across to the xiphoid process. A similar incision is then made on the opposite side of the body. From the xiphoid process, the incision is carried downwards to the symphysis pubis. (3) Modified "Y" shaped incision: An incision in midline from suprasternal notch to symphysis pubis. The incision extends from suprasternal notch over the clavicle to its centre on both sides and then passes upwards over the neck behind the ear (Fig. 5-1).

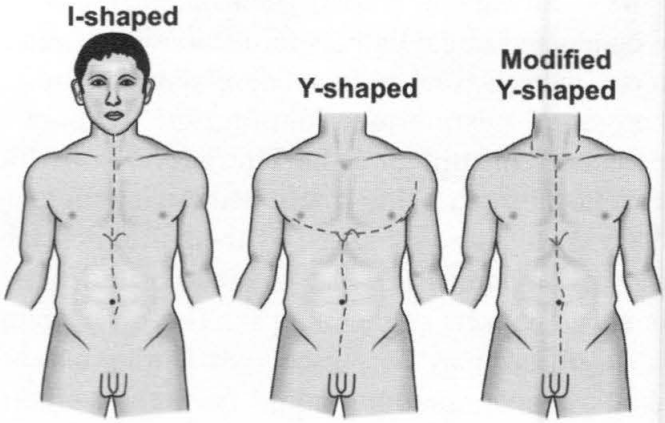


Fig. (5-1). Primary skin incisions.

INTERNAL EXAMINATION

OPENING OF BODY CAVITIES: ABDOMEN: The recti muscles of the abdomen are divided about 5 cm above symphysis. A small cut is made in the fascia big enough to admit the left index and middle fingers, palmar surfaces up. The fingers are separated in a V (to protect the underlying structures) and a sharp-bladed knife is inserted between them and the peritoneum is cut up to the xiphoid. The condition of the abdominal cavity and organs is observed before anything is disturbed or altered, to find out if there is any blood, pus or fluid in the cavity or perforation or damage to any organ. Note abnormalities and position of abdominal organs, adhesions, old operations, pathologic processes, injuries and height of diaphragm in relation to the ribs. The peritoneum is examined for adhesions, congestion, inflammation or exudate.

CHEST: PNEUMOTHORAX: Cases of pneumothorax are demonstrated before the chest wall is opened. (1) A pocket is dissected on the affected side between the chest wall and the skin, and is filled with water, and the wall is punctured with the knife under the water. If air under pressure is present, it will bubble out of the opening through the water. (2) A wide-bore (16-gauge) needle attached to a 50 ml syringe without the plunger is introduced into s.c. tissue over an intercostal space and the syringe is then filled with water. The needle is pushed to enter the pleural cavity. If air is present it will bubble out through the water. (3) Chest X-ray.

The muscles of the chest are dissected away. Chest is opened by cutting the costal cartilages near the costal end with a cartilage knife. In old persons where the rib cartilages are calcified, a pair of rib shears or hand-saw are used. Then disarticulate the sternoclavicular joint on each side.

AIR EMBOLISM: If air embolism is suspected, the head should be opened first, and the surface vessels of the brain examined for gas bubbles. Air embolism can be demonstrated by: (1) Before handling the thoracic organs, the pericardium is opened, heart is lifted upwards and the apex is cut with a knife. The left ventricle is filled with frothy blood, if air is present in sufficient quantity to cause death. (2) If the right ventricle contains air, the heart will float in water. (3) The pericardial sac is filled with water and the heart is punctured with a scalpel and twisted a few times. Bubbles of air will escape if air is present. (4) A wide-bore needle attached to a 50 ml syringe filled with water is inserted into the right ventricle. If air is present it will bubble out through the water. (5) Air in inferior vena cava can be demonstrated by puncturing it under water, and looking for escape of bubbles of gas. (6) Chest X-ray.

Method of removal of organs: (1) Virchow's technique: Organs are removed one by one. Cranial cavity is exposed first, followed by thoracic, cervical and abdominal organs. Spinal cord is removed from the back. (2) **Rokitansky's technique:** It involves in situ dissection in part, combined with *en block* removal. It is preferred choice in patients with highly transmissible diseases, such as HIV, hepatitis B. (3) **Letulle's technique:** Cervical, thoracic, abdominal and pelvic organs are removed *en masse* and dissected as organ block. (4) **Ghon's technique:** Cervical, thoracic, abdominal and urogenital system are removed as organ blocks. Neuronal system is removed as another block.

NECK STRUCTURES: A large blunt-pointed scissors is used to cut open the oesophagus from the posterior surface up to the cardiac end of the stomach. Note for the presence of any capsules, tablets, powders, etc., which should be preserved. The larynx, trachea and bronchi are examined by cutting them open from the posterior surface. The presence of blood, mucus, foreign bodies, vomited matter, tumours, inflammation, mucopus, etc., in them should be noted. The thyroid is removed and examined. The carotid arteries must be examined for the presence of thrombosis particularly at the bifurcation near the base of the skull. The hyoid bone, thyroid and cricoid cartilages are examined.

LUNGS: Place the lungs with the anterior surfaces uppermost and open the pulmonary artery, and continue into the lung tissue as far as small scissors will allow one to go. Look for thrombi, emboli and atherosclerosis. Trace the course of pulmonary veins into the lung, looking for evidence of thrombosis. To separate the lungs, the long-bladed knife is placed blunt edge upwards under the hilum of each lung and turned around, so that the sharp edge is upward. Then with a short sawing motion, the hilum is completely cut through. The lung is held on the upper surface by the left hand (or by an interposed sponge) and the organ cut across from apex to base with the large brain knife, held parallel to the board. This produces an anteroposterior slice. The lungs are examined for consolidation, oedema, emphysema, atelectasis, congestion, Tardieu spots, emboli, etc.

HEART: It is held at the apex and lifted upwards and pulmonary vessels, superior and inferior vena cavae, and the ascending aorta are cut as far away as possible from the base of the heart. **The pulmonary**

arteries should be palpated before they are cut, and looked for an embolus when they are incised. The pericardium is examined and incised and any blood or fluid in the pericardium is noted. **It is opened in the direction of the flow of blood** (inflow-outflow method), with the enterotome (Fig. 5-2). The right atrium is cut between the openings of superior and inferior vena cavae. The enterotome introduced into the right atrium, cuts through the tricuspid orifice, and opens the right ventricle along the lateral margin. The enterotome is introduced into the right ventricle close to the apex, and the conus pulmonalis and pulmonary valve are cut along the interventricular septum in the anterior wall of the right ventricle. The incision should extend into the pulmonary trunks and the left pulmonary artery. The left atrium is cut between the openings of the pulmonary veins. Then, the left atrium is cut along its lateral wall. The incision extends through the mitral orifice and passes along lateral margin of the left ventricle up to the apex. The next incision extends from the apex along the interventricular septum into the aorta, opening the aortic valve. Both auricular appendages should be examined for the presence of thrombi. The heart should be weighed, and measurements of the circumference of valves, and of thickness of right and left ventricle should be taken.

Coronary arteries: The coronary arteries are examined by making serial cross sections along the entire course of the major vessels about two to three mm apart, using a scalpel. This method demonstrates narrowing of the vessel and any antemortem thrombus in its lumen without danger of dislodging it (Plate 1-5). The muscle of the right and left ventricles is incised in a plane parallel to epicardial and endocardial surfaces, which reveals infarction and fibrosis most clearly.

Subendocardial Haemorrhages: The haemorrhages are seen in the left ventricle, on the left side of the interventricular septum and on the opposing papillary muscles and adjacent columnae carneae. The haemorrhages are flame-shaped, confluent and tend to occur in one continuous sheet rather than patches (Plate 1-4). When the bleeding is severe, it may raise the endocardium into a flat blister. They are seen: (1) after sudden severe hypotension due to severe loss of blood or from shock, (2) after intracranial damage, such as head injury, cerebral oedema, surgical

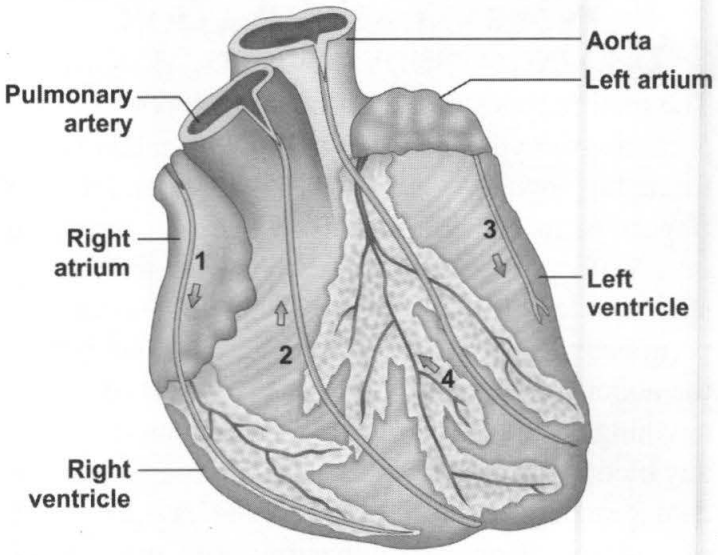


Fig. (5-2). Incisions into the isolated heart.

craniotomy, or tumours, (3) death from ectopic pregnancy, ruptured uterus, antepartum or postpartum haemorrhage, abortion, (4) various types of poisoning, especially arsenic.

POSTMORTEM CLOTS: Two types are seen. (1) When blood clots rapidly, a soft, lumpy, uniformly dark-red, slippery, moist clot is produced ("black currant-jelly"). (2) When red cells sediment before blood coagulates, the red cells produce a clot similar to the first type. Above this a pale or bright yellow layer of serum and fibrin is seen ("chicken fat"). Usually, a mixture of the two types of clot is seen. Postmortem clots are moist, smooth, rubbery, homogeneous, loosely or not at all attached to the underlying wall and there are no lines of Zahn. Postmortem fibrinous clots in heart are known as cardiac polyps.

STOMACH: The stomach is removed after applying double ligatures at each end and is placed in a clean dish. **It is opened along its greater curvature, from the cardiac to pyloric end.** Note the size of pyloric ring with a finger and open the duodenum along the anterior wall. The contents are examined for nature of any food which may be present and its state of digestion, smell, colour, character and also for the presence of foreign or suspicious matter. The mucous membrane is examined for the presence of congestion, haemorrhage, ulceration or other abnormal conditions.

INTESTINES: Examine the small and large intestines for colour, consistency of wall, adhesions, serosal surface, herniae or other abnormalities. The small intestine is opened along the line of mesenteric attachment and the large intestine along the anterior

taenia. They are examined for inflammation, congestion, erosion, ulcers, perforation, etc. The contents are also examined.

LIVER: It is removed by itself or attached to the stomach and the duodenum, when bile ducts are to be examined. Its weight, size, colour, consistency and the presence of any pathologic process or injury is noted. It is cut into two cm. slices thick, which run in the long axis.

SPLEEN: Note size, weight, consistency, condition of capsule, rupture, injuries or disease. It is sectioned in its long axis, and the character of parenchyma, follicles and septa noted.

PANCREAS: It is sliced by a series of cuts at right angles to the long axis. Examine for fat necrosis.

KIDNEYS: Note size and weight. The capsule is stripped and examined. The kidney is sectioned longitudinally through the convex border into the hilum so as to split in half and open the pelvis. The pelvis is examined for calculi and inflammation. The ureters are split by fine scissors.

BLADDER: It is opened from the fundus and incision carried into the urethra. The condition of the wall and amount and character of urine are noted.

PROSTATE: It is examined for enlargement or malignancy. Vertical cross-sections through the lateral and median lobes are made with knife.

TESTES: Incise the inguinal canal from the peritoneal aspect and pull out a loop of vas with finger. Free the vas to the internal inguinal ring. Push the testis up out of the scrotum with the right hand, and pull the vas with the left hand. The testis usually comes out without difficulty or damage. The testis and epididymis are held with the left hand and are cut longitudinally with knife. Note for the presence of any clotted blood inside the scrotum and around the testis.

FEMALE GENITALIA: The vagina and uterus are cut anteriorly or posteriorly up to the fundus. Two short incisions are made in the fundus from main longitudinal incision towards each cornu to expose endometrium. The ovaries are sectioned longitudinally and the tubes are cut across at intervals. If the uterus contains a foetus, its age should be determined.

HEAD: A wooden block is placed under the shoulders so that the neck is extended, and the head is fixed by a head rest, which should have a semicircular groove to hold the back of the neck.

Removal of skull cap: A coronal incision is made in the scalp, which starts from the mastoid process just behind one ear, and is carried over the vertex of the scalp to the back of the opposite ear (**intermastoidal incision**) (**Fig. 5-3**). The scalp is reflected forwards to the superciliary ridges, and backwards to a point just below to the occipital protuberance. The **saw-line is made in a slightly V-shaped direction** so that the skull cap will fit exactly back into the correct position (**Fig. 5-4**). The saw-line should go through the bones along a line extending horizontally on both sides from about the centre of the forehead to the base of the mastoid process, and from these latter points backwards and upwards to a point a little above the occipital protuberance. Care should be taken to avoid sawing through the meninges and brain. To avoid this, stop when saw meets little resistance. A chisel and hammer should not be used to loosen the skull completely. Skull cap is removed by gently inserting and twisting the chisel at various places through the cut (**Fig. 5-4**). The superior longitudinal sinus is opened along its length with a scalpel and carefully examined for an antemortem thrombus. The dura mater is grasped anteriorly with a forceps, and with scissors or scalpel the dura mater is divided from before backwards at the level of the skull division on both sides.

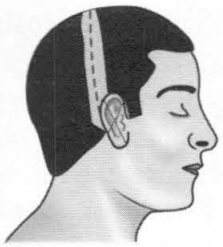


Fig. (5-3). Primary scalp incision.

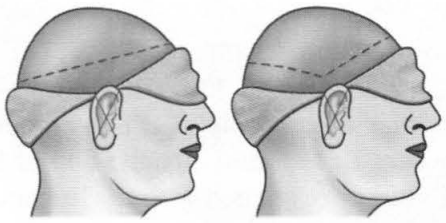


Fig. (5-4). Common method of removing the skull cap.

BRAIN: Cut the dura all along the line of cleavage of the skull and fold it back to midline. Free the falx cerebri from the cribriform plate and pull dura and falx backwards. The brain is removed by inserting the four fingers of left hand between frontal lobes and skull, and drawing frontal lobes backwards and cutting the vessels and nerves at the base. The tentorium is cut along the posterior border of the petrous bone. The knife is passed into occipital foramen and cervical cord, first cervical nerves and vertebral arteries are cut as far below as possible. The right hand grasps the cerebellum and the surface and base are examined for haemorrhage, injury or disease. The condition of the cerebral vessels, especially the vessels in the circle

of Willis is noted for the presence of arteriosclerosis, minute aneurysms, etc.

FIXATION OF BRAIN: For complete examination of the brain, it is fixed in 10% formalin for one week. To facilitate penetration of formalin, the lateral fissures are opened with the fingers which tear open the pia-arachnoid, and a long sagittal cut is made through the corpus callosum to allow formalin to pass into the ventricles. The brain can also be perfused with fixative through the arterial stumps before further fixation by immersion.

DISSECTION OF BRAIN: After fixation, the brain is placed upside down on a board. Remove the cerebellum with pons and medulla. Turn the cerebellum over and cut straight down through the vermis to expose the fourth ventricle. To expose the dentate nucleus, cut obliquely through each hemisphere. Whole of the stem is sectioned transversely at intervals of a few millimetres to demonstrate haemorrhages or other abnormalities. The examination of the cerebral hemispheres is made by a series of sagittal cuts downwards into the cerebrum, beginning at the frontal pole and passing backwards to the occiput, at intervals of about one cm. The slices should be moved aside in sequence and placed on the cutting board so that they can be identified consecutively later.

REMOVAL OF SPINAL CORD: It is **not examined routinely** unless there is an indication of disease or injury. The body is placed prone on the table, face downwards. A wooden block is placed under the chest, and the head is bent downwards. An incision is made on the back in midline extending from the occipital protuberance to the fourth lumbar vertebra. The muscles are dissected away from the top of the spinal column, noting their condition. The atlas is disarticulated. The laminae are sawed through the entire length of spine on each side of the spinous process, by means of an adjustable double blade saw, and are separated with the chisel. The dura is slit open along the midline with scissors and the presence of haemorrhage, inflammation, suppuration or tumour is noted. The nerves are cut from below up as they pass through spinal foramina. The cord is separated at the foramen magnum. The cord is sectioned transversely and serially. The vertebral column is examined for fractures or dislocation.

Cleaning and suturing of the cadaver: After completing the autopsy, the body cavities should be cleaned of blood, fluid, etc. The organs are replaced in the body and any excess space is packed with cotton, cloth, etc., especially in the pelvis and the throat, where blood tends to leak. The dissection flaps are brought close together and well sutured by using thin twine and a large curved needle. The skull should be filled with cotton or other absorbent material and the skull cap fitted in place. The scalp is pulled back over the calvarium and the scalp stitched with thin strong twine. The body should be washed with water, dried, and covered with clothes. **The body is handed over to the police constable accompanying it for disposal.**

AUTOPSY OF A CASE OF AIDS AND INFECTIOUS DISEASES

Highly infectious diseases transmitted by direct contact or contact with infected materials, clothing, discharges, vomit, etc., are cholera, rabies, tetanus, poliomyelitis, mumps, septicaemia, typhoid, tuberculosis, hepatitis B and C, AIDS, diphtheria, CSF meningitis, smallpox, plague, tick-borne encephalitis, equine encephalomyelitis, T-cell lymphotropic viruses I and II. Body fluids considered to transmit HIV include blood, semen, vaginal secretions, CSF, peritoneal, pericardial, amniotic and synovial fluids. Workers who have exudative lesions or weeping dermatitis or external injury should not handle AIDS victims. Infection of AIDS can be acquired by transdermal inoculation through cuts and needle punctures. About 0.5% of individuals will become seropositive. Transmission rates from contaminated needle puncture or close contact are 10 to 30 times higher for serum hepatitis than for AIDS. Viable HIV has been recovered from blood samples up to 16 days after death.

UNIVERSAL WORK PRECAUTIONS: (1) All infected bodies should be wrapped and tied in double layer tough plastic bag, with a red colour tag mentioning "Biologically Hazardous". The label should mention the name, age, sex, registration number, etc. (2) Proper protective clothing: Full sleeves overalls instead of simple surgical gowns, waterproof surgical apron, head cap, face mask, goggles if eye glasses are not worn, double gloves (heavy autopsy gloves over surgical gloves), waterproof rubber gumboots of knee length. (3) Avoid accidental pricks and cuts from needles, scalpels, etc. (4) If a cut is made in the rubber gloves or needle injury occurs they should be removed immediately and replaced with new ones. (5) Specimens for laboratory examination should be properly labelled and filled with 10% formalin solution and should be handled with gloved hands. (6) Used instruments should be dipped in 2% glutaraldehyde (cidox) for half-an-

hour, washed with soap or detergent and water, dried and then rinsed in methylated spirit and air dried. (7) All soiled gauze and cotton, etc. should be collected in a double plastic bag for incineration. (8) Laundry material, such as aprons, towels, etc., should be soaked in 1% bleach for half-an-hour, washed with detergent and hot water, and autoclaved. (9) Small spatters and spills of blood and other body fluids can be wiped up with disposable tissues or towels which are discarded in special biohazard bags and properly disposed. The autopsy table and floor should be cleaned with 1% bleach solution, followed by washing with soap and water. (10) Disinfectants: 1:10 dilution of common household bleach or a freshly prepared sodium hypochlorite solution is recommended. Liquid chemical germicides commonly used in health care facilities and laboratories are effective against HIV. (11) In case of accidental injuries or cuts with sharp instruments (contaminated with blood or body fluids or not), while working on a body, the wound should be immediately disinfected. (12) After autopsy all body orifices should be packed and the body should be wrapped in double layer heavy plastic sheet bag and secured properly, so that there is no leakage. A tag should be attached for identification. (13) After completing autopsy, hands and face should be washed with soap and water and rinsed in 70% methylated spirit. (14) The body should be burnt or incinerated.

It is better to leave some organs in situ in the cadaver, rather than eviscerating *en masse.* Another method is to fix lungs and other organs whole, after removal rather than slicing them before fixation. If the recommended guidelines are adhered to, the dangers of performing autopsies are absolutely minimal.

THE AUTOPSY AND DISPOSAL OF RADIOACTIVE CORPSE: If the amount of radioactivity is less than 5 millicuries, no extraprecautions are necessary. If the body contains between 5 and 30 millicuries of radioactive material, the doctor must wear heavy rubber gloves, plastic aprons, plastic shoe covers and spectacles to reduce contamination. Instruments with long handles should be used during the autopsy. Organs and fluids that are most radioactive should be removed first and placed in covered glass jars, labelled and examined for radioactivity from time to time. Fluid of the pleural and peritoneal cavity should be flushed copiously with running water and disposed off directly into the sewer. Contaminated wearing dress should be thoroughly cleaned with soap and water, and stored for suitable decay of the radioactive material before being sent to laundry. Instruments can be brought to a safe level by soaking them in water with soap or a detergent. Contamination of the floor of the autopsy room should be avoided. Spilled fluid should be cleaned with dry disposable water, held with forceps and put into a suitable vessel. If the radiation level is high, a team may be required to complete the autopsy. Special organs may be removed first, and detailed dissection done away from the body, or after a period of cold storage, or fixation to permit radioactive decay. If the body contains more than 300 millicuries of activity after autopsy, it should be embalmed in the hospital morgue. The presence of a cardiac pacemaker must be recorded, especially if it is one which might contain mercury or a radioactive substance, as explosions in crematoria from the heating of mercury batteries have proved hazardous.

ENDOSCOPIC TECHNIQUE OF POSTMORTEM DIAGNOSIS: The method requires the use of rigid Hopkins-endoscope of 4 mm and 8 mm diameters with 0°, 90°, and 130° view angles having a light source from fibreglass cable. The endoscope is fitted with zoom lens camera for photographic documentation.

Sinuses, fundus, external auditory meatus, larynx, etc. can be directly visualised. Bullet tracts in the body can be traced accurately. Hepatic, splenic and diaphragmatic injuries and intraperitoneal and thoracic haemorrhages can be visualised. Natural orifices can be clearly examined.

VIRTUAL AUTOPSY (VIRTOPSY): It is a non-invasive technique of examining dead bodies to find out the cause of death. It is a combination of CT and MR imaging. CT images give information about morbid anatomical findings and MR imaging demonstrates soft tissue injury, organ trauma, state of blood vessels, tissues and bones. MR spectroscopy measures metabolites formed due to decomposition, which help to estimate time since death. Two dimensional and three dimensional imaging is done with multislice CT. Emphysema, air embolism, pneumothorax, hyperbaric trauma and decompression effects can be better appreciated. When an injury is caused by a weapon, using a computer software, virtual model of an injury with the 3D image of a simulation can be created by using a similar weapon. The track of the projectile inside the brain or other internal organs with haemorrhage and damage to tissues can be viewed with CT scanning.

PSYCHOLOGICAL AUTOPSY: Psychological autopsy is a set of postmortem investigative procedures (gathering all the relevant information from the past life of the victim of suicide); that help ascertain and evaluate the role that physical

and psychological factors play in the death of an individual, thus to determine the manner of death to as high a degree of certainty as possible. In some cases of suicide, police reports may be incomplete as source of information. Description of scene of death, position of body, evidence at the scene, such as weapons, poison and notes, etc., are necessary. **Equivocal deaths are those that do not indicate the manner of death, e.g., they were not witnessed or involved conflicting data. It involves systematic collection of psychological data (mental state) through structured interviews of the deceased's family members, friends, co-workers, employers and fellow students, who had dealt with the deceased are helpful,** to reconstruct the habits of the victim as regard to his background, habits, character, personality traits, general lifestyle and method of death. It is most commonly introduced in those involving custodial care-taking (police custody and prisons), and those of contested life insurance claims. They have also been introduced in criminal cases (suicide or homicide), workers compensation (unsafe job conditions), product manufacturer's (depressants) and professional negligence (proximate cause of suicide). The psychological autopsy cannot validly inform an expert that a suicide definitely occurred, as the information gathered may be biased, but it **significantly improves manner of death determination. The procedure also has therapeutic value, leading to bereavement counselling, the relieving of guilt feelings, and the answer to other questions.**

PRESERVATION OF VISCERA IN CASES OF SUSPECTED POISONING: The following must be preserved in all fatal cases of suspected poisoning:

(1) **Stomach** and its contents.

(2) **The upper part of small intestine** (about 30 cm length) and its contents.

(3) **Liver** not less than 0.5 kg.

(4) **One kidney** or half of each kidney, as one kidney may be dysfunctional.

(5) **Blood** 30 ml. It should be collected from a peripheral site; either from axillary, jugular or femoral veins. Blood should not be collected from heart cavities, inferior vena cava or portal or hepatic veins. When blood samples are collected for volatile substances, small glass bottles should be used, which should be filled to the top to avoid loss of volatile substances into the head space and stored at 4°C.

(6) **Urine** 30 ml. It can be obtained by catheter or suprapubic puncture with syringe and long needle before autopsy, or by making an incision on the anterior surface of the bladder during autopsy.

For grouping a piece of filter paper or clean cotton gauze can be used to soak up some blood and dried.

As most poisons are taken orally, the poison is most likely to be present in the stomach and intestinal contents and in their wall. After absorption all poisons pass through the liver, which is the major detoxicating organ and has the power of concentrating many poisons. The kidney being the organ of excretion contains large amounts of poison, which is excreted into the urine.

If the poison is only found in the contents of the stomach, and none in the solid viscera, doubt may occur about the actual cause of death. Poison found in liver, kidney and blood is proof of absorption. Therefore, it is important to keep the contents of the stomach and intestine in a separate bottle. **Poison found in urine, unless added with evil intention, is a proof of absorption and excretion.** If the poison is also found in the food or medicine preserved, this would be very strong additional evidence. **The urine contains the drug or poison in many cases. It is suitable for single direct spot tests. In delayed deaths, the toxic agent may be found in the urine, when none is found in the viscera.**

Preservatives: (1) **Saturated sodium chloride** solution. It is not used in poisoning from corrosive acids except phenol, alkalis, corrosive sublimate and aconite. (2) **Rectified spirit,** except in cases of suspected poisoning by (a) alcohol, (b) acetic acid, (c) phenol, (d) paraldehyde, (e) phosphorus, chloroform, ether, chloral hydrate, formic acid, formaldehyde, because the organic acids and paraldehyde are soluble in alcohol and the phosphorescence of phosphorus is diminished by alcohol. (3) A 10 mg/ml of sodium or potassium fluoride (prevents glycolysis, inhibits enzyme enolase and bacterial growth) and 3 mg. potassium oxalate should be used for preserving blood. Fluoride 10 mg for every one ml. should also be added to urine and vitreous humour if alcohol estimation is required, and also to samples for analysis for cocaine, cyanide and CO. In poisoning by oxalic acid, ethylene glycol, fluoride and CO, 30 mg. sodium citrate should be added for 10 ml. of blood. One ml

of concentrated hydrochloric acid or one g. of thymol or one g of sodium fluoride can be used for 100 ml of urine as preservative. Toluene is better.

Preservative is not necessary if (1) viscera can be analysed within 24 hours, (2) if samples can be kept in a refrigerator or in an ice box, (3) bone, hair, nails, (4) lung for detecting inhaled poisons.

N.B.: The **viscera should not be preserved in formaldehyde** because extraction of poison, especially non-volatile organic compounds, becomes difficult.

For grouping, a piece of filter paper or clean cotton gauze can be used to soak up some blood and dried.

INSTRUCTIONS FOR PRESERVATION AND DISPATCH OF VISCERA: (1) The stomach and its contents and the small intestine and its contents are preserved in one bottle, and the liver and kidney in another bottle. The blood and urine are preserved separately. The bottles should be clean, wide-mouthed, white and fitted with glass stoppers. (2) The stomach and intestines are opened before they are preserved. The liver and kidney are cut into small pieces so that they are well preserved. (3) **The quantity of preservative should be equal to or more than the viscera in bulk.** (4) **Only two-thirds of the capacity of the bottle should be filled with the viscera and preservative to avoid bursting of bottle** if gases of decomposition are formed. (5) The stoppers of bottles should be well-fitted, covered with a piece of cloth and tied by tape or string and the ends sealed. (6) The bottles should be labelled which should contain the name of the victim, autopsy number, the organs it contains, and the date and place of autopsy. (7) A sample of the preservative used, i.e., 10 ml. of rectified spirit or 25 g. of sodium chloride is separately kept in a bottle and sent for analysis, to exclude the possibility of any poison being present as a contaminant. (8) The sealed bottles containing the viscera and preservative are put into a box, which is locked and the lock is sealed. (9) A copy of the inquest report, postmortem report and the authorisation from the Magistrate are sent to the Forensic Science Laboratory along with the viscera. (10) The key of the box and a sample seal on a piece of paper corresponding to the seal used on bottles and lock are kept in an envelope, which is sealed and sent with viscera box. (11) The viscera box is handed over to the police constable after taking a receipt, who delivers it personally in the office of the Forensic Science Laboratory, after obtaining a receipt for the same.

Label to be Pasted on Viscera Bottle

Hospital: **Place:**

Cr. No: ..... **Date:** ..... **Police Station:** .....

Postmortem No. : **Date:**

Name of the deceased: **Age:** **Sex:**

Nature of specimen :

Preservative used: Rectified spirit/saturated saline/ sodium fluoride.

Signature of Medical Officer

Preservation of additional viscera: In certain cases of poisoning the following articles are preserved. (1) Brain: Alkaloids, organophosphorus compounds, opiates, CO, cyanide, barbiturates and volatile organic poisons. (2) Spinal cord: Strychnine. (3) Heart: Strychnine, digitalis, yellow oleander. (4) Lung: Gaseous poisons, hydrocyanic acid, alcohol, chloroform, etc. (5) Vitreous humour: Alcohol, chloroform, cocaine, morphine, tricyclic antidepressants, etc. (6) Bile: Narcotic drugs, cocaine, methadone, glutathione, barbiturates and some tranquilisers. (7) Skin: In deaths where hypodermic injection marks or areas where absorption of poison through the skin may have occurred, an area of ten cm radius about the site, with as much underlying fat and muscle as possible and the whole needle track is removed. (8) Bone: An 8 to 10 cm of the shaft of the femur is taken in cases of subacute or chronic poisoning by arsenic, antimony, thallium or radium. (9) Hair: An adequate sample of head hair should be removed with a tweezer to remove the roots, and tied in locks. (10) Nails: All the nails should be removed entirely. (11) Uterus: Uterus and appendages and upper part of vagina are preserved in cases of criminal abortion. Sticks and foreign bodies found in the genital tract are preserved separately. (12) Muscle: The muscles, especially of thigh, is often well preserved, even in advanced decomposition. In embalmed body, the best specimen may be skeletal muscle from the buttock.

Cause of death: After completing the postmortem examination, a complete but concise report should be written in duplicate using carbon papers. One copy is sent to the investigating officer and another copy is retained for future reference. The report should be sent as soon as possible. **A definite opinion should be given whenever possible, but if the cause of the death cannot be found out, it**

should be mentioned in the report. In such cases, viscera should be preserved, and histological and bacteriological examinations carried out. In suspected cases of poisoning, the opinion should be kept reserved until the report is received from the laboratory. The conclusion that death was caused by poison depends on evaluation of clinical, autopsy, toxicologic and circumstantial evidence. If the cause of death is not found on autopsy, the opinion as to the cause of death should be given as "undetermined", and the manner of death as "unknown". When there are no positive postmortem findings, the cause of death can be inferred from the accurate observations by reliable witnesses, concerning the circumstances of death.

Autopsy of Decomposed Bodies: Even when the body shows advanced decomposition, a thorough examination may show a gross traumatic or pathological lesion. The skin though discoloured, may show the presence of a gross external injury, e.g., a bullet wound or a large incised wound. Fractures are easily detected. Gross pathological lesions may be found, e.g., valvular lesions of the heart.

Effacement or obliteration of identity: The identity of a dead body may be destroyed or obliterated by the following methods. (1) Purposive removal of the identifying features, e.g. fingerprints, tattoo marks, scars, moles, teeth, hair, etc., and articles of clothing. (2) Animals, e.g., rats, dogs, jackals and hyenas and birds, such as vultures may attack a dead body and mutilate it in a very short time, when the body is exposed in an open place. (3) Advanced putrefaction. (4) Burning or incineration. (5) Dismemberment and burying or throwing different parts in different places. (6) Dismemberment by moving vehicles like trains or by machinery. (7) Chemical destruction of the body in corrosive acids or alkalis. (8) Bomb explosions, which may disintegrate the body. **Biocremation or chemical hydrolysis:** The body is placed in a stainless steel cremation chamber (similar to pressure cooker). A mixture of 95% water and 5% potassium hydroxide is added. 150°C heat and 60 pounds of pressure a square inch is applied. The average adult body takes about 2 to 3 hours to complete a biocremation the sterile solution is recycled to the earth.

EXAMINATION OF MUTILATED BODIES OR FRAGMENTS: Mutilated bodies are those which are extensively disfigured, or in which a limb or a part is lost but the soft tissues, muscles and skin are attached to the bones. Sometimes, only a part of the body, such as head, trunk or a limb may be found.

(1) **Human or Animal:** This is easy if the head, trunk or limbs are available, but when pieces of muscle only are available without attached skin or viscera, it is very difficult. In such cases, definite opinion can be given by performing precipitin test or antiglobulin inhibition test, using blood, or any other soft tissue, if the tissue is not severely decomposed.

(2) **One or more Bodies:** This is determined by fitting together all separate parts. If there is no disparity or reduplication, and if the colour of the skin is same in all parts, they belong to one body.

(3) **Sex:** It can be determined if the head or trunk is available, from the presence and distribution or absence of hair, characters of the pelvis, and skull. It can also be determined from the recognition of prostatic or uterine tissue under a microscope which resist putrefaction, and are found even in advanced state of putrefaction.

(4) **Age:** Age can be determined from general development, skull, teeth and ossification of bones.

(5) **Identity:** It can be determined from finger prints, tattoo marks, scars, moles, hair, teeth, deformities, etc., articles of clothing and superimposition technique.

(6) **Stature:** It can be determined from measurement of long bones.

(7) **Manner of Separation of Parts:** This is determined by examining the margins of the parts, whether they had been cleanly cut, sawn, hacked, lacerated, disarticulated at the joints or bitten by animals.

(8) **Time Since Death:** The probable time since death may be determined from the stage of decomposition of parts.

(9) **Cause of Death:** The cause of death can be made out if there is evidence of fatal injury to some vital organ or large blood vessels, or marks of burning, or deep cuts, or fractures of bones, especially of the skull or the cervical vertebrae, hyoid bone, or of several ribs.

(10) **Antemortem or Postmortem:** This may be determined by examination of the margins of the parts for evidence of vital reaction.

EXAMINATION OF BONES

Forensic anthropology is that branch of physical anthropology, which, for forensic purposes, deals with the identification of skeletonised remains known to be or suspected of being human. Sometimes, bones are found disposed off in a jungle, in the open, in the ditches or rubbish dumps, etc., or may be found while digging foundations for buildings, or skeleton may be exhumed.

General Description: Keep the bones in anatomic arrangement and draw a skeletal chart indicating which bones are present. A complete list of all the bones sent for examination should be prepared and photographs of all the bones are taken. The sand, dust or earth present on the bones is removed with brushes and wooden picks and scrapers. Note the attachments of soft tissues to bones if any, and their stage of putrefaction. Note whether the bones are moist and humid or dry and their smell. The bones are then washed by brushing with lukewarm water, and are placed under shelter to dry slowly. If soft tissues are attached, the bones are boiled in water for 5 to 6 hours in the case of young bones, and for 12 hours or more in the case of adult bones. With this, the soft parts, including tendon attachments and the periosteum can be removed easily with an ordinary scrubbing brush.

(1) **Human or Animal:** It is easy when whole skeleton or complete bones are available. Difficulties arise when there is marked fragmentation, burnt bones and with smaller bones of some animals, such as digits, metacarpal and metatarsal bones. **Bones of the hand and wrist of bears may be confused with human hands.** If the bone is fairly fresh and some of the blood constituents are still present, the precipitin test is useful. **If the bones are fresh, DNA analysis can be done from the marrow cells for identification of species.** Serological tests are not useful in case of bones not having extractable plasma proteins (5 to 15 years old bones), and burnt and cremated bones. **Microscopic structure is also useful.** Human and animal bones can also be distinguished by **chemical analysis of bone ash**.

(2) **One or more Individuals:** This can be determined by reconstructing the skeleton. If there is no disproportion in the size of various bones or reduplication, articulation is correct and if the age, sex and race of all the bones is same, they belong to one individual. **If mixing of bones from more than one skeleton is suspected, they can be separated by the use of a shortwave ultraviolet lamp by the difference in the colour emissions.**

(3) **Sex: Recognisable sex differences are not present before puberty.** After puberty, the sex can be determined by examination of the pelvis, skull, diameter of head of femur and humerus, and measurements of femur, tibia, humerus and radius.

(4) **Age at Death:** It can be determined from examination of teeth, ossific centres, amount of wear and tear in teeth, length of long bones, epiphyseal union, pubic symphysis, closure of skull sutures, cortical resorption, bony lipping, osteoporosis, calcification, osteoarthritic changes, etc. After the completion of bony union, the exact age cannot be determined. In case of broken bones, a rough estimate of the age can be made by measuring the external diameter of the long bones at midshaft.

(5) **Race:** There are certain racial differences in the skeleton, chiefly in the skull and face measurements, teeth and lower extremities. Foetal age can be determined by measuring the length of the ossified portions of the long bones.

(6) **Stature:** When the skeleton is incomplete or severely disintegrated, the stature may be calculated by applying mathematical formulae to the length of the long bones. Long bones must be measured by means of osteometric board; measurement by the use of tapes or calipers are not accurate. **Pearson's formulae** were used for a number of years. **Trotter and Gleser's formulae** are more satisfactory. **The principle of these formulae is to measure the length of long bone and multiply it with a given factor, and then adding a fixed factor.** The formulae are different for dry bones and wet bones, for white persons and Negroes, and for males and females. Formulae for stature are not valid for children, giants or dwarfs.

Weight-bearing long bones are used for applying these formulae. Femur and tibia give more accurate results than humerus or radius. Right side bones are normally measured in the dry state without cartilage. The average stature obtained from measurement of more than one long bone gives a more accurate result than when calculated from a single bone. The error may be up to two-and-half cm.

Steele (1970) has devised a method for estimating stature from fragmented pieces of major long bones. In the absence of long bones, adult stature can be calculated from the articular length of the five metacarpals.

A simple means of estimating stature assumes that the length of the humerus is 20%, the tibia 22%, the femur 27%, and the spine 35%, of the stature.

Osteometric Board: This has a rectangular base with a ruler incorporated along one of its long sides. An upright is fixed at one end of the board and a second one slides along the board. The bone is placed with one of its ends against the fixed upright and the movable upright is brought up to the other end of the bone. The distance between the uprights is then shown on the ruler. Hepburn osteometric board modified by Trevor is commonly used **(Fig. 5-5)**.

If osteometric board is not available, the bones should be measured on a flat bench with the maximum lengths taken between two vertical, parallel boards placed in contact with the bone ends. If the bones are covered with articular cartilage, subtract for radius and humerus 3 mm each, tibia 5 mm and femur 7 mm before applying the formulae.

(7) **Identification:** Identity may be established from teeth, disease and deformities of the bone, old healed or healing fractures, orthopaedic surgical procedures, regional atrophy, spinal deformities, flat feet, supernumerary ribs, congenital defects, etc., and by superimposition technique using the skull. If previous X-rays are available, it may be possible to make positive identification on the basis of contours and cancellous patterns of various bones. Dental charts and dental radiographs are also useful.

(8) **Nature of Injury:** The ends of the long bones should be examined carefully to find out if they have been cut by sharp cutting instruments, or sawn or bitten by animals. If the body disintegrates naturally, articular surfaces are smooth. Postmortem conditions may cause fractures and fragmentation due to continued and repeated freezing and thawing and from the pressures of shifting soil weight.

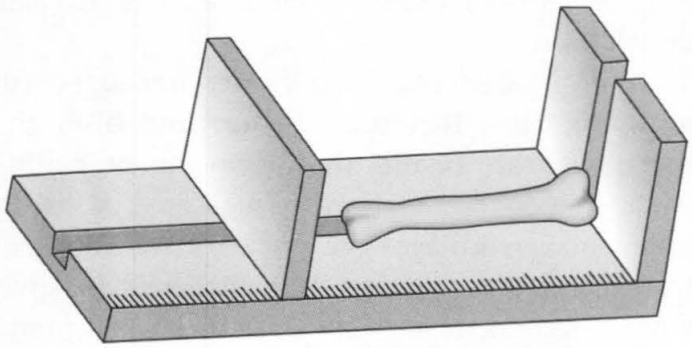


Fig. (5.5). Osteometric board.

Burnt bones: Heat makes the bones brittle, so that breakage may occur during collection. The shape of burnt bone is preserved, but it becomes powder when pressed between the fingers. **In a burnt skull, the cracking often has a circular pattern with sharp edges which look like cuts.** Unprotected bone when burnt undergoes charring, cracking (usually transverse), splintering and may burn to ashes, whereas bone embedded in thick, soft tissue shows the molten or guttered condition characteristic of fusion by heat. A bone burnt in the open is white, but when burnt in a closed fire, black or ash-grey.

(9) **Time since Death:** It is difficult to determine the exact time since death. **It depends upon the circumstances under which the bones have been found, e.g., burial and cold weather will diminish decomposition. After the soft tissues disappear, pieces of cartilage and ligaments remain attached to bone for three to four months.** A fairly recent bone is slightly greasy to the touch and is heavy. Traces of marrow and periosteum may remain in, or attached to the bones for several months. Odour is a good indication of relatively recent death. After the bones have lost the covering tissue and the odour of decomposition, the bones still appear fresh. Ancient bones tend to be dry, brittle, chalky and the marrow cavity is dry, free of fat, and often contains particles of earth or sand. In cases of a fracture, the callus should be examined after cutting it longitudinally to ascertain the age of fracture. With decomposition, the bones lose organic matter and weight.

(10) **Cause of Death:** The cause of death cannot be made out unless there is evidence of fracture or injuries which usually cause death, e.g., factures of skull bones, upper cervical vertebrae, hyoid bone, several ribs, or marks of deep cuts in long bones or marks of burning. The bones should be examined for firearm injuries or any disease, e.g., caries or necrosis. The type of the weapon can be known from the type and depth of the cut in the bone. Metallic poisons, e.g., arsenic, antimony, lead or mercury can be found in bones long after death. **Arsenic can be detected even in burnt bones.**

EXHUMATION

Exhumation is the legal digging out of an already buried body from the grave. There is no time limit for exhumation in India.

Autopsies are performed on exhumed bodies: (1) **In criminal cases**, such as homicide, suspected homicide disguised as suicide or other types of death, suspicious poisoning, death as a result of criminal abortion and criminal negligence. (2) **In civil cases**, such as accidental death claim, insurance, workmen's compensation claim, survivorship and inheritance claims or disputed identity.

Authorisation: The body is exhumed only when there is a written order from the Executive Magistrate.

Procedure: The body is exhumed under the supervision of a medical officer in the presence of a police officer and Magistrate. **The grave site should be positively identified with identifying features,** such as location of burial plot, the headstone, and from the persons present at the time of burial. **It should be conducted in natural light.** If the grave is in an open place with lot of spectators, the area should be screened off. Notes should be made about the condition of the soil, water content, and vegetable growth. After the dirt has been removed from above and around the corpse, it should be photographed in the position in which it was found. The condition of the burial clothes, and the surface of the body should be noted. In cases of suspected mineral poisoning, samples of the earth (about half kg.) in actual contact with the body and also from above, below and from each side should be collected. Any fluid or debris in the coffin should also be collected. A portion of the coffin and burial clothes must be removed in order to exclude any possibility of contamination from external sources. **The body should be identified by the close relatives and friends.** All personal effects, clothing, hair, nails, etc., should be picked up for examination.

P.M. Examination: Disinfectants should not be sprinkled on the body. If the body has been buried recently, the postmortem examination is conducted in the usual manner. Various artifacts have to be interpreted correctly. In much putrefied bodies, an attempt should be made to establish the identity. All the viscera should be preserved for chemical analysis. If the body is reduced to skeleton, the bones should be examined.

Second Autopsy: The interpretation of the findings of a second autopsy performed on a previously autopsied exhumed body is difficult due to the various artifacts of burial and exhumation, and serious alterations resulting from the first autopsy. The findings should be noted in great detail, whether the findings are confirmatory or different from the result of first autopsy. It is possible that valuable results may be obtained. **Even if no new information is obtained from the second autopsy, it will help in putting an end to rumours or suspicions.**

CHAPTER

6 Death and Its Cause

Thanatology deals with death in all its aspects.

Types: (1) somatic, systemic or clinical, and (2) molecular or cellular.

SOMATIC DEATH: It is the complete and irreversible stoppage of the circulation, respiration and brain functions (Bishop's triad of life), but there is no legal definition of death. The question of death is important in resuscitation and organ transplantation. As long as circulation of oxygenated blood is maintained to the brain stem, life exists. A person who cannot survive upon withdrawal of artificial maintenance is dead.

Organ transplantation: The success of a homograft mainly depends upon the type of tissue involved and the rapidity of its removal after circulation has stopped in the donor. Cornea can be removed from the dead body within six hours, skin in 24 hours, bone in 48 hours and blood vessels within 72 hours for transplantation. Kidneys, heart, lungs, pancreas, intestines and liver must be obtained soon after circulation has stopped as they deteriorate rapidly.

BRAIN DEATH: Harvard criteria: (1) **Unreceptivity and unresponsivity:** Total unawareness to externally applied stimuli and inner need and complete unresponsiveness to even the most intense painful stimuli. (2) **No movements:** No spontaneous muscular movements in response to stimuli, such as pain, touch, sound or light for a period of at least one hour. (3) **Apnoea:** Absence of spontaneous breathing for at least one hour, and when patient is on ventilator, the total absence of spontaneous breathing may be established by turning off the respirator for 3 minutes and observing whether there is any effort on the part of the subject to breathe spontaneously. (4) **Absence of elicitable reflexes:** Irreversible coma with abolition of central nervous system activity is evidenced in part by the absence of elicitable reflexes. The pupils are fixed and dilated and do not respond to a direct source of bright light. Ocular movements and blinking are absent. There is no evidence of postural activity. Corneal and pharyngeal reflexes are also absent. Stretch tendon reflexes also cannot be elicited. (5) **Isoelectric EEG:** It has confirmatory value.

All these tests should be repeated after 24 hours with no change. Further it is stressed that the patient be declared dead before any effort is made to take him off the ventilator, if he is then on a ventilator.

There are two distinct schools of diagnosing death: (i) French and English schools, that is similar to Harvard. (ii) Austro-German school that includes Harvard criteria and bilateral serial angiography of internal carotid and vertebral artery criteria. A negative angiogram for more than 15 minutes proves death.

Diagnosis of Brainstem Death: Exclusions: (1) Where the patient may be under the effects of drugs, e.g., therapeutic drugs or overdoses. (2) Where the core temperature of the body is below 35°C. (3) Where the patient is suffering from severe metabolic or endocrine disturbances which may lead to severe but reversible coma, e.g., diabetes.

Preconditions of diagnosis: (1) Patient must be deeply comatose. (2) Patient must be maintained on a ventilator. (3) Cause of the coma must be known.

Personnel who should perform the tests: (1) Brainstem death tests must be performed by two medical practitioners. (2) Doctors involved should be experts in this field. Under no circumstances are brainstem death tests performed by transplant surgeons. (3) At least one of the doctors should be of consultant status. Junior doctors are not permitted to perform these tests. (4) Each doctor should perform the tests twice.

Tests to be performed: The diagnosis of brainstem death is established by testing the function of cranial nerves which pass through the brainstem. If there is no response to these tests, the brainstem is considered to be irreversibly dead.

When two doctors have performed these tests twice with negative results, the patient is pronounced dead and a death certificate can be issued.

Tissue and Organ Transplantation: (1) **Homologous donation** means grafting of the tissue from one part of the body to another in the same patient, such as skin or bone. (2) **Live donation** includes blood and bone marrow transfusion. Live organ donation includes kidney and parts of the liver. (3) **Cadaveric donation:** most organs must be obtained while the donor heart is still beating to improve chances of success.

Xenograft is grafting of animal tissue into humans, which has limited success. **Zoonoses** are transmission of animal diseases.

VEGETATIVE STATE: In this the patient breathes spontaneously, has a stable circulation and shows cycles of eye opening and closing which may simulate sleep and waking, but is unaware of the self and the environment. It is usually seen in patients with diffuse, bilateral cerebral hemisphere disturbance with an intact brainstem, though it can occur with damage to the most rostal part of the brainstem.

Molecular Death: It means, the death of cells and tissues individually, which takes place usually one to two hours after the stoppage of the vital functions. Molecular death occurs piecemeal. Nervous tissues die rapidly, the vital centres of the brain in about 5 minutes, but the muscles live up to one to two hours.

Agonal period is the time between a lethal occurrence and death.

Modes of Death: There are three modes of death, depending on whether death begins in one or other of the three systems, irrespective of what the remote cause of death may be. These modes are: (1) Coma, (2) Syncope, and (3) Asphyxia.

COMA

Coma is the insensibility which involves the central portion of the brain stem. Coma is a clinical symptom and is not a cause of death.

Causes: (1) **Compression of the brain,** e.g., effusion of blood on or in the brain, inflammation, abscess or neoplasm of brain. (2) **Drugs:** opium, hypnotics, cocaine, alcohol, anesthetics, cyanide, atropine, phenol, oxalic acid, CO, etc. (3) **Metabolic disorders** and infections: Uraemia, cholaemia, eclampsia, diabetes, pneumonia, infectious fevers, heat stroke, etc. (4) **Other causes:** Embolism and thrombosis in the cerebral vessels, epilepsy, hysteria, etc.

Autopsy: Injuries or disease of the brain may be present as noted in the causes of coma. The lungs, brain and the meninges are congested.

SYNCOPE

Syncope is sudden stoppage of action of the heart, which may prove fatal. This term is also not used as a cause of death. Syncope or fainting is due to vasovagal attacks resulting from reflex parasympathetic stimulation. Syncope is caused by reflex bradycardia or asystole, or by reflex splanchnic vasodilation. Due to the acute reflex circulatory changes, blood pressure falls suddenly causing cerebral anaemia and rapid unconsciousness. Recovery is common.

Causes: (1) Anaemia due to sudden and excessive haemorrhage. (2) Asthenia from deficient power of heart muscle, as in fatty degeneration of the heart, myocardial infarction and certain poisons. (3) Vagal inhibition. (4) Exhausting diseases.

Autopsy: The heart is contracted and the chambers are empty when death has occurred from anaemia, but chambers contain blood when death occurs due to asthenia. The lungs, brain and abdominal organs are usually pale and the capillaries are congested.

ASPHYXIA

Asphyxia is a condition caused by interference with respiration or due to lack of oxygen in respired air, due to which the organs and tissues are deprived of oxygen (together with failure to eliminate CO_2), **which may cause unconsciousness and death. The term asphyxia indicates a mode of dying, rather than a cause of death.** Nervous tissues are affected first by deficiency of oxygen, and their functions are disturbed even by mild oxygen lack. The neurons of the cerebral cortex will die in 3 to 7 minutes of complete oxygen deprivation, and the other nerve cells remain alive for a little longer time. **Subnormal oxygen in the blood supply to the brain causes rapid unconsciousness. The rule of thumb is: breathing stops within 20 seconds of cardiac arrest, and heart stops within 20 minutes**

of stopping of breathing. If the heart functions for several minutes after stoppage of breathing, the weight of the lungs may increase to 450 to 500 g.

ANOXIA: It means lack of oxygen. It may be: **(1) Anoxic anoxia:** In this type, oxygen cannot reach the blood, because of lack of oxygen in the lungs. This occurs: (a) from breathing in a contaminated atmosphere, e.g., from exposure to the gases in wells and tanks, or from exposure to sewer gas, (b) from mechanical interference with the passage of air into or down the respiratory tract, e.g., in smothering, choking, hanging, strangulation, traumatic asphyxia, drowning, and certain forms of acute poisoning. **(2) Anaemic anoxia:** In this type, oxygen carrying capacity of the blood is reduced, e.g., acute massive haemorrhage, acute poisoning by carbon monoxide, chlorates, nitrates, and coaltar derivatives. **(3) Stagnant anoxia:** In this type, impaired circulation results in a reduction of oxygen delivery to the tissues, e.g., heart failure, embolism, and shock. **(4) Histotoxic anoxia:** In this type, the enzymatic processes by which the oxygen in the blood is used by the tissues are blocked, e.g., acute cyanide poisoning. Anoxic anoxia is usually known as asphyxia, or mechanical asphyxia. **These four types of anoxia ultimately lead to cardiac failure and death.**

Types and Causes: (1) Mechanical: In this the air-passages are blocked mechanically. (a) Closure of the external respiratory orifices, as in smothering. (b) Closure of the air-passages by external pressure on the neck, as in hanging, strangulation, and throttling, etc. (c) Closure of the air-passages by the impaction of foreign bodies in the larynx or pharynx, as in choking. (d) Prevention of entry of air due to the air-passages being filled with fluid, as in drowning. (e) External compression of the chest and abdominal walls interfering with respiratory movements, as in traumatic asphyxia.

(2) Pathological: In this entry of oxygen to the lungs is prevented by disease of the upper respiratory tract or of the lungs, e.g., bronchitis, acute oedema of the glottis, laryngeal spasm, tumours and abscesses.

(3) Toxic: Poisonous substances prevent the use of oxygen. (a) The capacity of haemoglobin to bind oxygen is reduced, e.g., poisoning by CO. (b) The enzymatic processes by which the oxygen in the blood is used by the tissues are blocked, e.g., cyanides. (c) Respiratory centre may be paralysed in poisoning by opium, barbiturates, strychnine, etc. (d) The muscles of respiration may be paralysed by poisoning by gelsemium.

(4) Environmental: (a) Insufficiency of oxygen in the inspired air, e.g., enclosed places, trapping in a disused refrigerator or trunk. (b) Exposure to irrespirable gases in the atmosphere, e.g., sewer gas, carbon monoxide, carbon dioxide. (c) Exposure to high altitude.

(5) Traumatic: (a) Pulmonary embolism from femoral vein thrombosis, due to an injury to the lower limb. (b) Pulmonary fat embolism from fracture of long bones. (c) Pulmonary air embolism from an incised wound of internal jugular vein. (d) Bilateral pneumothorax from injuries to the chest wall or lungs.

(6) Postural asphyxia: This is seen where an unconscious or stuporous person, either from alcohol, drugs or disease, lies with the upper half of the body lower than the remainder.

(7) Iatrogenic: It is associated with anaesthesia.

Pathology: Compression of the neck: Reduction in oxygen causes capillary dilation which is followed by stasis of blood in the dilated capillaries and venules, which produces capillo-venous engorgement. This blood stasis causes congestion of organs, and venous return to the heart is diminished leading to anoxia, which causes capillary dilation and the vicious cycle goes on **(Fig. 6-1)**.

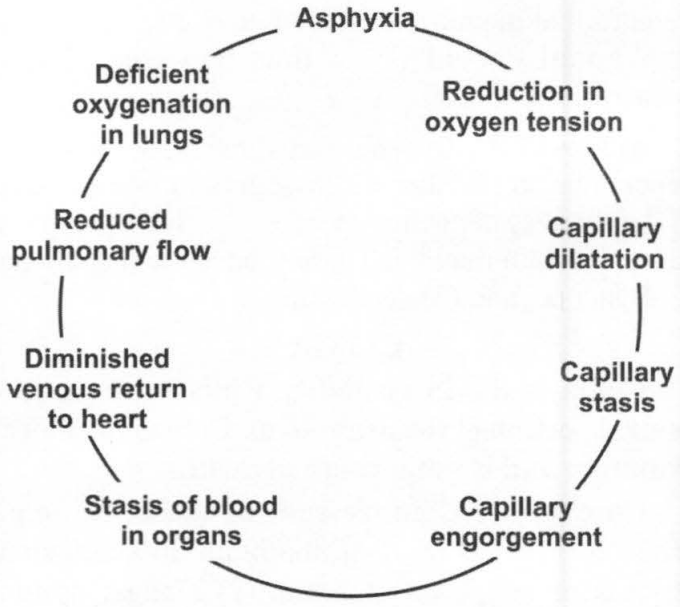


Fig. (6-1). Vicious cycle of asphyxia.

Petechial haemorrhages: They are caused due to raised venous pressure from impaired venous return resulting in overdistension and rupture of capillaries and venules, and not due to hypoxia of the vessel walls **(Figs 6-2 and 6-3)**. A minimum of 15 to 30 seconds is required to produce congestion and petechiae. Petechiae vary in size from 0.1 to 2 mm. If larger than this they are called **ecchymoses.** Sometimes, large haemorrhages are seen on upper chest and back of the body. Petechiae are seen in the skin, sclerae, conjunctivae **(Figs 6-2 and 6-3 and Plate 1-6)**, outer and inner surfaces of the eyelids, and on the mucosal surfaces in the mouth. The haemorrhages may be found in the substance of the viscera, but they are readily seen in serous membranes, particularly in the visceral pleura, in the interlobar fissures and around the hilum and pericardium. In the brain, they are seen in the white matter **(Plate 2-7)** and there may be larger patches of bleeding in the subarachnoid space. The same mechanism often produces profuse petechiae and ecchymoses under the scalp. Petechiae are rarely seen in the parietal pleura and peritoneum except in haemorrhagic diathesis.

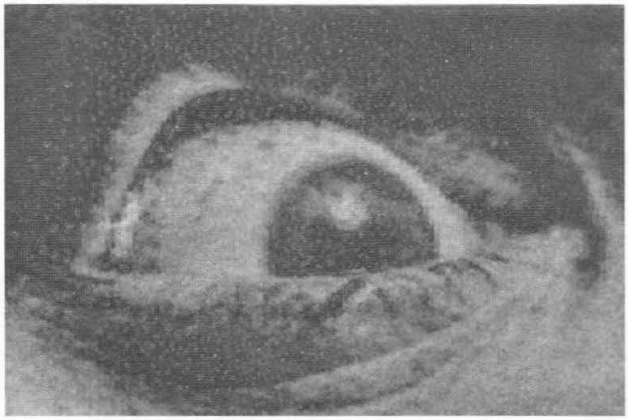


Fig. (6-2). Subconjunctival petechial haemorrhages.

Fig. (6-3). Tardieu spots on foot and leg in a case of hanging.

Congestion: It occurs due to obstructed venous return. When the neck is compressed, face, lips and tongue become swollen and reddened. The colour change of congestion is usually darkened by the onset of cyanosis. Internal organs are also congested.

Oedema: If the hypoxia continues, oedema of the tissues develops. Oedema of the lungs is common. Generalised oedema is not prominent. The increased capillary and venous permeability causes transudation of fluid into tissues. **Congestion and oedema are non-specific and result due to obstructed venous return.**

Cyanosis: Cyanosis (bluish colour of the skin, mucous membranes and the internal organs), especially lungs, liver, spleen, kidneys and meninges, results from diminished oxygen tension in the blood and an increase of reduced haemoglobin. **The cyanotic colour of the blood will not be detected unless there is at least 5 g./100 ml. of reduced haemoglobin present in the capillary blood.** Oxygenated blood is bright red. It becomes purplish-blue when oxygen is given up. About 24 hours after death, oxygen will be dissociated from the blood and cyanosis will appear in any dead body.

Asphyxial Stigmata: Asphyxia is not a pathological entity, and cannot be clearly recognised from morbid anatomical findings alone. **The triad of (1) cyanosis, (2) facial, palpebral, bulbar, subpleural and subepicardial petechiae, (3) visceral congestion, are consistent with but not diagnostic of asphyxia from anoxic anoxia.** Reliable local indications of fatal obstructing trauma must be demonstrated, to establish that death occurred from mechanical asphyxia.

SIGNS AND SYMPTOMS: (1) STAGE OF DYSPNOEA: The excess of carbon dioxide in the blood stimulates the respiratory centre. The respiratory movements become increased in rate and amplitude, blood pressure is increased, pulse rate increases and there is slight cyanosis.

(2) STAGE OF CONVULSIONS: The effort to breathe is mostly expiratory, the face is deeply congested, blood pressure is increased, pulse rate increases, and veins in the neck become swollen. Frequent convulsions occur which stop as the victim becomes insensible and the reflexes are abolished.

(3) STAGE OF EXHAUSTION: The respiratory centre is paralysed. There is complete insensibility, reflexes are lost and the pupils are widely dilated. The breathing is gasping, mostly inspiratory with long intervals between the gasps. B.P. falls, muscles relax, respiration ceases and death occurs. The pulse is imperceptible, but the heart may continue to beat for some minutes after respirations have ceased. The three stages last for 3 to 5 minutes before death occurs.

Postmortem Fluidity of the Blood: Shortly after death the blood is usually fluid, and when it is removed from the body it undergoes spontaneous coagulation. If the autopsy is done a few hours after death, the blood may be partly clotted and partly fluid. In most deaths from asphyxia, the blood is fluid and incoagulable. The postmortem fluidity of the blood is due to presence of fibrinolysins that occur in rapid deaths, possibly by high agonal levels of catecholamines. It is not characteristic of asphyxia or any cause of death. Under certain conditions, the fibrinolysins may be so active that fibrin is destroyed as rapidly as it is produced, and postmortem clots never develop in the vessels. In other cases thrombi are formed, but they undergo lysis. The blood is fluid in CO poisoning, in rapid death from asphyxia, with large doses of anticoagulants, retained abortion, puerperal sepsis, amniotic fluid embolism, and certain cases of septicaemia.

Postmortem Appearances: External: Postmortem hypostasis is well developed. The **face** is either pale in slow asphyxia or distorted, congested, often cyanosed and purple, and sometimes swollen and oedematous. **Ears and fingernails are bluish**. The **eyes** are prominent, the conjunctivae are congested and the pupils are dilated. The **tongue** is protruded in most cases, and frothy and bloody mucus escapes from the mouth and nostrils. Postmortem discharge of semen from the meatus is common in any type of death not only in asphyxia. **Petechial haemorrhages, known as Tardieu spots are produced by simple mechanical obstruction to the venous return of blood from the parts, resulting in acute rise in venous pressure and over-distension and rupture of thin-walled peripheral venules and capillaries, especially in lax unsupported tissues,** such as forehead, circumoral skin, the eyelids, conjunctivae, sclerae, and skin behind the ears. They are most marked where for mechanical reasons, capillary congestion is most prominent. **Their distribution lies above the level of obstruction.** They appear commonly in the scalp, eyebrows and face in hanging and strangulation, and in the zone above the level of compression in traumatic asphyxia. They may occur in many non-asphyxial states. In some asphyxial deaths petechiae are seldom found.

Internal: The **blood** is fluid and dark, because of increased amount of CO_2. The large veins are full of blood. Vessels may burst in the eardrum and in the nose causing bleeding. The larynx and trachea are usually congested and contain a varying amount of slightly frothy mucus. The **lungs** are dark and purple. If the back-pressure persists, there is exudation of serous or serosanguineous fluid in the alveoli, producing oedema. Some of the marginal portions of the lungs may show emphysematous change. The abdominal viscera show marked venous congestion. The brain is often congested. The cranial sinuses are usually filled with dark blood. Dilatation of heart chambers on right side and fluidity of blood in deaths due to asphyxia are absolete and should be disregarded. **Tardieu spots are numerous where the capillaries are least firmly supported,** as in subconjunctival tissues and under the pleural and pericardial membranes, but they can appear almost anywhere if the degree of congestion and cyanosis is sufficient. **Tardieu spots are usually round, dark and well-defined haemorrhages varying in size from a pin's head to 2 mm.** They may occur as isolated minute haemorrhages **(Fig. 6-3)** or present in large numbers, and at times combine to form patches of red colour, especially at the back of the heart. In many cases of fetal hypoxia, asphyxia signs are absent, such as sudden blockage of trachea by food, plastic bag pulled over the head or when victim enters a space devoid of oxygen.

Natural diseases: Asphyxial signs are found in many kinds of natural disease. Petechial haemorrhages beneath the pleura and pericardium are seen in many natural diseases and in normal postmortem hypostasis, especially where the mode of death was congestive as in many types of natural heart disease, apart from asphyxia. They may develop after death. As such petechiae are highly unreliable indicators of an asphyxial process. Haemorrhages in the skin are seen in bacterial endocarditis, meningococcal septicaemia, blood dyscrasias, especially purpura and haemophilia, and also in deaths from coronary thrombosis, acute heart failure, secondary shock, and rapid anoxia. These conditions produce relatively large haemorrhages which tend to combine. Their

distribution is general, whereas Tardieu spots are present above the level of obstruction. In these cases, death occurs rapidly due to cardiac arrest.

Vomiting: Asphyxia causes vomiting due to medullary suboxia, due to which the air-passages may be filled at the end of asphyxial event by the inhaled vomit. Probably due to lack of normal protective mechanisms of air passages at or about the time of death. This finding especially in infants, should not be assumed to be the cause of asphyxia; it is more likely to be the result of asphyxia. About 20 to 25% of all individuals aspirate food agonally, whatever may be the cause of death.

Variations: There are variations in the intensity of asphyxial signs. If during the process of asphyxia, heart failure occurs before respiratory failure, the asphyxial signs may be less marked. **Sometimes, the findings are not sufficient to use an accurate term, and the cause of death has to be given within a broad framework, such as "consistent with asphyxia".**

In the presence of **putrefaction**, mechanical asphyxia is indicated by presence of petechial haemorrhages under the eyelids, conjunctivae, sclerae, and facial skin.

The cause of death is the disease or injury, responsible for starting the sequence of events, and which produce death.

The manner of death indicates the circumstances under which the person died. It is established from the personal and family history, circumstantial information, the scene of death, witnesses of the event, information from family members and others, and by the autopsy findings. **If death occurs exclusively from disease, the manner of death is natural.** If death occurs exclusively by injury or is hastened due to injury in a person suffering from natural disease, the manner of death is unnatural or violent. Violence may be suicidal, homicidal, accidental or of undetermined or unexplained origin.

The mechanism of death is the physiological or biochemical disturbance produced by the cause of death which is incompatible with life, e.g., shock, sepsis, toxaemia, severe metabolic acidosis and alkalosis, ventricular fibrillation, respiratory paralysis, etc.

NEGATIVE AUTOPSY: When gross and microscopic examination, toxicological analyses and laboratory investigations fail to reveal a cause of death, the autopsy is considered to be negative. Two to five per cent of all autopsies are negative. A negative autopsy may be due to: (1) **Inadequate history:** Deaths from vagal inhibition, status epilepticus, hypersensitivity reaction, laryngeal spasm in drowning, etc., may not show any anatomical findings. (2) **Inadequate external examination:** The presence of fresh and old needle marks may be missed on cursory examination in a drug addict. Death from snake bites and insect bites cannot be explained unless the bite marks are identified. The burn may be missed in electrocution. (3) **Inadequate or improper internal examination:** Air emobolism and pneumothorax are often missed. (4) **Insufficient laboratory examinations.** (5) **Lack of toxicological analysis.** (6) **Lack of training of doctor.**

The obscure causes are: Where no lesion is found at autopsy, or if a lesion is found it is of a minimal or indefinite nature. **(1) Natural diseases:** (a) With obscure or few macroscopic findings, (b) death precipitated by emotion, work stress, and (c) with functional failure, such as epilepsy, paroxysmal fibrillation.

(2) Biochemical disturbances: (a) Uraemia, diabetes, potassium deficiency, and (b) respiratory pigment disorders, such as anaemic anoxia, porphyria.

(3) Endocrine dysfunction: Adrenal insufficiency, and (b) thyrotoxicosis or myxoedema.

(4) Concealed trauma: (a) Cerebral concussion, (b) self-reduced neck injury, (c) blunt injury to the heart, and (d) reflex vagal inhibition.

(5) Poisoning: Without macroscopic change: (a) Delayed suboxic or narcotic poisoning; (b) anaesthetic overdosage or maladministration; (c) neurotoxic or cytotoxic poisons, and (d) plant poisoning.

(6) Miscellaneous: (a) Allergy. (b) drug idiosyncrasy, etc.

Non-medical persons believe that the cause of death can always be determined by autopsy. The police may press the doctor for giving a positive statement with regard to the cause of death. In such cases, the doctor should admit his inability to give a positive opinion. Such obscure cases require clinical and laboratory investigations and interview with persons who had observed the deceased before he died, to know the signs and symptoms shown by the deceased before his death. Laboratory investigations may be bacteriological, histological, biochemical,

and toxicological. The possible presence of infectious, malignant, occupational, and other diseases are also excluded. In the absence of positive findings from these sources, a careful assessment of possible functional causes of death must be made, before any cause of death is given as undetermined. Even in these frustrating cases, **the negative morbid anatomical and chemical examination is of great value in excluding injuries or poisons, which may have been alleged to have caused or played some part in the death. Though the pathologist cannot establish the cause of death, he can exclude many conditions which have been incorrectly involved as having some aetiologic role or importance in the death.**

INHIBITION OF THE HEART: (Vagal inhibition; vaso-vagal shock; reflex inhibition; nervous apoplexy or instantaneous physiological death): Sudden death occurring within seconds or a minute or two due to minor trauma, or relatively simple and harmless peripheral stimulation is caused by vagal inhibition.

Mechanism: Pressure on the baroreceptors situated in the carotid sheaths, carotid sinuses, and the carotid body (located in the internal carotid artery just above the bifurcation of common carotid artery) causes an increase in blood pressure in these sinuses with resultant slowing of the heart rate, dilatation of blood vessels and a fall in blood pressure. In normal persons, pressure on the carotid sinus causes minimal effects with a decrease in heart rate of less than six beats per minute, and only a slight reduction (<10 mm Hg) in blood pressure. **Some individuals show marked hypersensitivity to stimulation of the carotid sinuses, characterised by bradycardia and cardiac arrhythmias ranging from ventricular arrhythmias to cardiac arrest.** Stimulation of the carotid sinus baroreceptors causes impulses to pass via Hering's nerve to the afferent fibres of the glossopharyngeal nerve (9th cranial nerve); these in turn link in the brainstem to the nucleus of the vagus nerve (10th cranial nerve). Parasympathetic efferent impulses then pass to the heart via the cardiac branches of the vagus nerve. Stimulation of the fibres causes a profound bradycardia. This reflex arc is independent of the main motor and sensory nerve

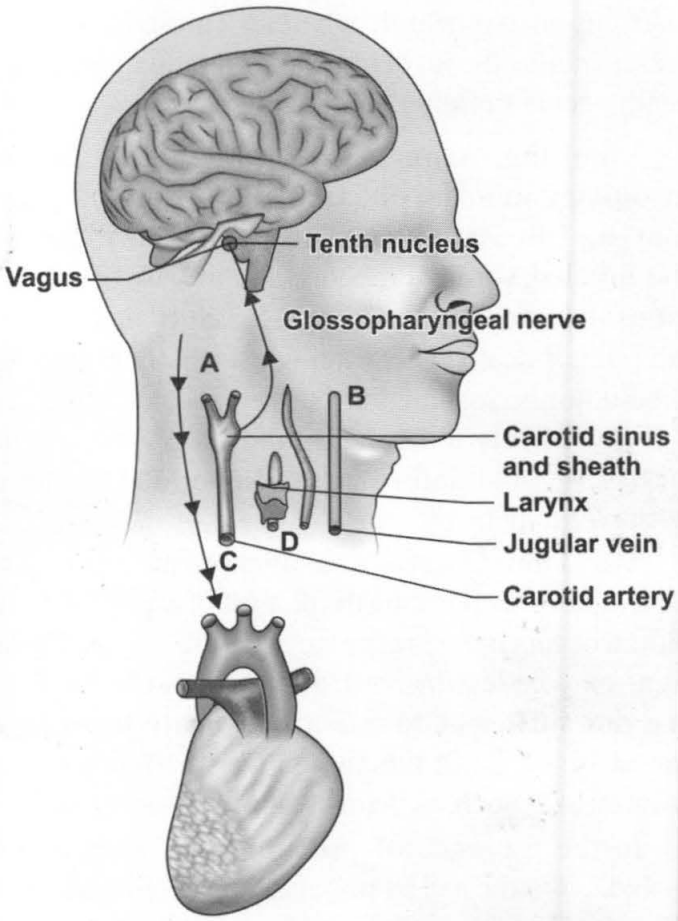


Fig. (6-4). The coarse distribution of the receptor system in vagal inhibition reflexes.

pathways. There is wide network of sensory nerves in the skin, pharynx, glottis, pleura, peritoneum covering viscera or extending into the spermatic cord, cervix, urethra, perineum and coeliac plexus (receptor areas). Afferent fibres from these tissues pass into the lateral tracts of the spinal cord, effect local reflex connections over several segments and also pass to the brain **(Fig. 6-4)**.

Causes: (1) The commonest cause of such inhibition is pressure on the neck, particularly on the carotid sinuses as in hanging or strangulation. (2) Unexpected blows to the larynx, chest, abdomen or testicles. (3) Impaction of food in larynx. (4) Unexpected inhalation of fluid into the upper respiratory tract. (5) Sudden immersion of body in cold water. (6) Insertion of an instrument into the bronchus, uterus, bladder or rectum. (7) Sudden distension of hollow muscular organs, e.g., during attempts at criminal abortion, when instruments are passed through the cervix or fluids are injected into the uterus. (8) Extensive injuries to the spine

or other parts of the body. (9) Sudden evacuation of pathological fluids, e.g., ascitic or pleural. (10) Puncture of a pleural cavity, usually for induction of a pneumothorax.

Variation: There is great variation in individual susceptibility. Death from inhibition is accidental and initiated by microtrauma. The stimulus should be sudden and abnormal for the reflex to occur. The reflex is exaggerated by a high state of emotional tension, and also any condition which lowers voluntary cerebral control of reflex responses, such as a mild alcoholic intoxication, a degree of hypoxia or partial narcosis due to incomplete anaesthesia.

Autopsy: When death results from inhibition, **there are no characteristic postmortem appearances.** The cause of death can be inferred only by exclusion of other pathological conditions and from the accurate observations by reliable witnesses concerning the circumstances of death.

SUDDEN DEATH

Death is said to be sudden or unexpected when a person not known to have been suffering from any dangerous disease, injury or poisoning is found dead, or dies within 24 hours after the onset of terminal illness. The incidence is approximately 10% of all deaths. It may occur at any age.

Causes: (I) Diseases of the Cardiovascular System (45 to 50%): (1) Coronary atherosclerosis with coronary thrombosis. (2) Coronary atherosclerosis with haemorrhage in the wall causing occlusion of the lumen. (3) Coronary artery disease (narrowing and obliteration of the lumen by atherosclerosis). (4) Occlusion of the ostium of the coronary artery associated with atherosclerosis or syphilitic aortitis. (5) Coronary artery embolism. (6) Rupture of the fresh myocardial infarct. (7) Acute pericarditis. (8) Acute endocarditis. (9) Acute myocarditis. (10) Pulmonary embolism. (11) Systemic embolism occurring in bacterial endocarditis. (12) Fatty degeneration of the heart. (13) Valvular lesions: aortic stenosis, aortic regurgitation, mitral stenosis, rupture of the chordae, ball-valve thrombus. (14) Cardiomyopathies, alcoholic myopathy, asymmetrical hypertrophy of the heart. (15) Congenital heart disease in the newborn. (16) Lesions of the conducting system: fibrosis, necrosis. (17) Angina pectoris. (18) Arterial hypertension with atherosclerosis. (19) Spontaneous rupture of aorta. (20) Rupture of aortic or other aneurysm.

(II) Respiratory System (15 to 23%): (1) Lobar pneumonia. (2) Bronchitis and bronchopneumonia. (3) Acute oedema of the lungs. (4) Lung abscess. (5) Massive collapse of the lung. (6) Influenza. (7) Diphtheria. (8) Acute oedema of the glottis. (9) Neoplasm of the bronchus. (10) Impaction of foreign body in the larynx and regurgitation of stomach contents into air-passages and bronchioles. (11) Bronchial asthma. (12) Pleural effusion. (13) Pneumothorax caused by rupture of emphysematous bleb. (14) Rupture of blood vessel in pulmonary tuberculosis with cavitation. (15) Pulmonary embolism and infarction. (16) Air embolism.

(III) Central Nervous System (10 to 18%): (1) Cerebral haemorrhage. (2) Cerebellar haemorrhage. (3) Pontine haemorrhage. (4) Subarachnoid haemorrhage. (5) Brain abscess. (6) Brain tumour. (7) Meningitis. (8) Acute polioencephalitis. (9) Cysts of third or fourth ventricle. (10) Epilepsy. (11) Cerebral thrombosis and embolism. (12) Carotid artery thrombosis.

(IV) Alimentary System (6 to 8%): (1) Haemorrhage into the gastrointestinal tract from peptic ulcer, oesophageal varices, cancer oesophagus, etc. (2) Perforation of ulcers, e.g., peptic, typhoid, amoebic or malignant. (3) Twisting and intussusception of the bowel. (4) Paralytic ileus. (5) Intestinal obstruction. (6) Strangulated hernia. (7) Appendicitis. (8) Bursting of the liver abscess. (9) Rupture of enlarged spleen. (10) Obstructive cholecystitis. (11) Acute haemorrhagic pancreatitis.

(V) Genitourinary System (3 to 5%): (1) Chronic nephritis. (2) Nephrolithiasis. (3) Obstructive hydronephrosis and pyonephrosis. (4) Tuberculosis of the kidney. (5) Tumours of the kidney or bladder. (6) Rupture of ectopic pregnancy. (7) Toxaemia of pregnancy. (8) Uterine haemorrhage due to fibroids. (9) Twisting of ovary, ovarian cyst or fibroid tumour. (10) Cancer vulva eroding femoral vessel.

(VI) Miscellaneous (5 to 10%): (1) Diabetes mellitus. (2) Hyperthyroidism. (3) Addison's disease. (4) Blood dyscrasias. (5) Mismatched blood transfusion. (6) Haemochromatosis. (7) Cerebral malaria. (8) Shock due to emotional excitement. (9) Reflex vagal inhibition. (10) Anaphylaxis due to drugs.

CHAPTER

7 Postmortem Changes

SIGNS OF DEATH AND CHANGES FOLLOWING DEATH

Forensic taphonomy is the study and interpretation of postmortem processes of human remains, i.e., **the history of a body following death.** A knowledge of the signs of death help to differentiate death from suspended animation. The changes which take place after death are helpful in estimation of the approximate time of death. The signs of death appear in the following order.

Immediate (somatic death): (1) Insensibility and loss of voluntary power. (2) Cessation of respiration. (3) Cessation of circulation.

Early (cellular death): (4) Pallor and loss of elasticity of skin. (5) Changes in the eye. (6) Primary flaccidity of muscles. (7) Cooling of the body. (8) Postmortem lividity. (9) Rigor mortis.

Late (decomposition and decay): (10) Putrefaction. (11) Adipocere. (12) Mummification.

Insensibility and Loss of Movement: It is the earliest sign of death, but it can lead to error if precautions are not taken. They are found in cases of prolonged fainting attack, vagal inhibition, epilepsy, trance, catalepsy, narcosis, drowning, electrocution, etc.

Cessation of Respiration: This must be complete and continuous. The stethoscope is placed over the upper portions of the lungs and larynx, where the faintest breath-sounds can be heard. **Complete stoppage of respiration for more than 4 to 5 minutes usually causes death.** Respiration may stop for a very short period without death occurring in (1) as a purely voluntary act, (2) Cheyne-Stokes breathing, (3) drowning, and (4) newborn infants.

Feather test, mirror test and Winslow's test are of historical importance only.

Cessation of Circulation: The stethoscope is placed over the precordial area, where the heart-beat can be heard. Under normal conditions, **stoppage of heartbeat for more than four to five minutes is accepted as evidence of death.**

Magnus test, Icard's test, diaphanous test, fingernail, cut and heat test are of historical importance only.

SUSPENDED ANIMATION (apparent death)**: In this condition, signs of life are not found as the functions are interrupted for some time or are reduced to minimum. However, life continues and resuscitation is successful in such cases.** The metabolic rate is so reduced that the requirement of individual cell for oxygen is satisfied through the use of oxygen dissolved in the body fluids. In freezing of the body or in severe drug poisoning of the brain, the activity of brain can completely stop, and in some cases start again.

Types: (1) **Voluntary: Practitioners of yoga can pass into a trance, death-like in character.** (2) **Involuntary** suspension of animation lasting from a few seconds to half-an-hour or more may be found in newborn infants, drowning, electrocution, sunstroke, cholera, narcotic poisoning, after anaesthesia, cerebral concussion, shock, insanity, etc. This condition can be revived by cardiac massage or electric stimulator, and artificial respiration.

All postmortem changes are dependent on ambient temperature. High temperature accelerates the changes.

CHANGES IN THE SKIN: Skin becomes pale, ashy-white and loses elasticity within a few minutes of death.

CHANGES IN THE EYE: (1) Loss of corneal reflex: This is found in all cases of deep insensibility and therefore not a reliable sign of death. **(2) Opacity of the cornea:** This may occur in certain diseases (cholera, wasting diseases), before death. The opacity is due to drying and is delayed for about 2 hours, if the lids are closed after death. If the eyelids are open for a few hours after death, a film of cell

debris and mucus forms two yellow triangles on the sclera, each at side of the iris, with base towards the margin of cornea and apex towards medial or lateral canthus of the eye, which become brown and then black called **"tache noir"** within a few hours, upon which dust settles and the surface becomes wrinkled **(Plate 2-8)**. **(3) Flaccidity of the eyeball:** The eyes look sunken and become flaccid due to reduction of intraocular tension. **(4) Pupils:** Soon after death pupils are slightly dilated, because of relaxation of muscles of the iris. Later, they are constricted with the onset of rigor mortis of the constrictor muscles, and evaporation of fluid. **(5) Retinal vessels:** Fragmentation or segmentation (trucking) of the blood columns in the retinal vessels appear within minutes after death, and persists for about an hour. The retina is pale for the first 2 hours. At about 6 hours, the disc outline is hazy and becomes blurred in seven to ten hours. These changes are of little practical value. **(6) Chemical changes:** A steady rise in the potassium values occur in the vitreous humour after death up to 100 hours.

COOLING OF THE BODY (ALGOR MORTIS)

The cooling of the body (chill of death) after death is a complex process, which does not occur at the same rate throughout the body.

Loss of body heat: The body will not cool according to Newton's law. Energy and heat production stop after somatic death, which results in fall of body temperature. **The postmortem rate of heat production by anaerobic glycolysis is very low. Exchange of heat between the core and surface of the body occurs only by conduction.** The body cools more rapidly on the surface and more slowly in the interior. **For about half to one hour after death, the rectal temperature falls little or not at all** (postmortem temperature plateau). This is followed by a **linear rate of cooling (between 0.4 and 0.6°C. per hour) for the next 12 to 16 hours.** Then it gradually becomes slower and when the temperature is within about 4°C. of the environment, rate of cooling becomes very slow. The human body rarely reaches the ambient (atmospheric) temperature unless the latter is at or near freezing,

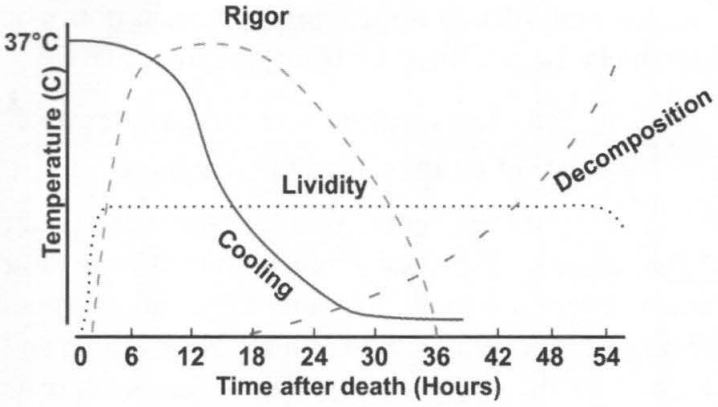


Fig. (7-1). Chart showing the major changes to estimate time since death.

probably because enzymes and bacterial action starts during early decomposition. **The curve of cooling is sigmoid or inverted S-shaped in pattern (Fig. 7-1).** Conductive heat exchange occurs due to the temperature difference between the body and surroundings, e.g., clothing, covering, air, water, etc. **At non-contact areas, heat exchange occurs by convectional mechanism, which exceeds that of contact surface. The body heat is mostly lost by conduction and convection.** Only a small fraction of heat is lost by evaporation of fluid from the skin. In serious illness, circulation begins to fail before death and hands and feet become cooler than the rest of the body; this coolness gradually extends towards the trunk. In sudden death, the cooling starts after death.

Measurement of temperature: A laboratory thermometer 25 cm. long, with a range of 0 to 50°C., which can be read in single degrees is used. (1) **The rectum is the ideal place to record temperature except in cases of sodomy.** The thermometer should be inserted 8 to 10 cm., and left there for two minutes. (2) The temperature can also be recorded by making a small midline opening into the peritoneal cavity in contact with the inferior surface of the liver, and inserting the thermometer under the liver. (3) The external auditory meatus or the nasal passages also can be used to record temperature. (4) Electronic thermocouple is better to record temperature. The time of this reading is recorded, and temperature of environment is recorded at the same time. Reading should be made at intervals in order to obtain the rate of fall of temperature.

A rough idea of approximate time in hours of death can be obtained by using the formula:

$$\frac{\text{Normal body temperature – rectal temperature}}{\text{Rate of temperature fall per hour}}$$

Variations in rectal temperature: It cannot be assumed that the body temperature is normal at death. There are individual and daily variations up to 1 to 1.5°C., being lowest in the early morning and highest in the late evening. Rectal temperature is between 36.5 and 37.5°C. and is about 0.6° to 1°C. higher than oral. During sleep the rectal temperature is half to one°C. lower. Low temperature occurs in cases of collapse, congestive cardiac failure, massive haemorrhage, secondary shock, etc. Marked rise in temperature occurs in cases of fat or air embolism, drug reactions, thyrotoxicosis, psychotic (emotional) stress, administration of neuroleptic medication, intoxication with heroin and cocaine, certain infections, heat stroke and in pontine haemorrhage. Exercise or struggle before death may raise the rectal temperature up to 1.5° to 2°C.

Factors Affecting Rate of Cooling: (1) The difference in temperature between the body and medium: The temperature fall is rapid when the difference between body and air temperature is great. In India, during summer, the temperature of the environment may be higher than that of the body temperature, and as such the cooling is very slow. **In tropical climates the heat loss is roughly 0.4°C to 0.6°C and in temperate countries 1°C per hour. (2) The build of the cadaver:** The rate of heat loss is proportional to the weight of the body to its surface area. Thus, children and old people cool more rapidly than adults. **(3) The physique of the cadaver:** Fat is a bad conductor of heat. Fat bodies cool slowly and lean bodies rapidly. **(4) The environment of the body:** A body kept in a well-ventilated room will cool more rapidly than one in a closed room. Moist air is a better conductor of heat than dry air, so that cooling is more rapid in humid atmosphere than in dry atmosphere. Cooling in water is rapid because, water is a far better conductor of heat. **Cooling in still water is about twice as fast as in air, and in flowing water, it is about three times as fast.** Bodies buried in earth cool rapidly than those in air but more slowly than those in water. **(5) Covering on or around the body:** The rate of cooling is slow when the body is clothed, as clothes are bad conductors of heat. A bedspread cloth covering may at least halve the rate of cooling.

Because of the above factors an accurate formula cannot be devised to define rate of heat loss. The rectal temperature of an average-sized naked body reaches that of environment in about twenty hours. **If the body is exposed to a source of heat for a few hours shortly after death, its temperature will rise.** A body in zero weather may undergo freezing and become stony hard.

Medicolegal Importance: It helps in the estimation of the time of death, which is not reliable. **The importance of temperature estimates apply only to cold and temperate climates.** In tropical zones, the postmortem fall in temperature may be minimal.

Postmortem Caloricity: In this condition, the temperature of the body remains raised for about first two hours after death. This occurs (1) when the regulation of heat production has been severely disturbed before death, as in sunstroke and in some nervous disorders, (2) when there has been great increase in heat production in the muscles due to convulsions, as in tetanus and strychnine poisoning, etc., and (3) when there has been excessive bacterial activity, as in septicaemic condition, cholera and other fevers.

POSTMORTEM HYPOSTASIS

This is bluish-purple or purplish-red (due to deoxyhaemoglobin) discolouration which appears under the skin in the most superficial layers of the dermis (rete mucosum) of the dependent parts of the body after death, due to capillo-venous distention. It is also called postmortem staining, subcutaneous hypostasis, livor mortis, cadaveric lividity, suggilations, vibices and darkening of death.

Mechanism: It is caused by the stoppage of circulation, the stagnation of blood in blood vessels and its tendency to sink by force of gravity. The blood tends to accumulate in the toneless capillaries and venules in the subcutaneous tissues of the dependent parts of the body. Filling of these vessels produces a bluish-purple colour to the adjacent skin. The upper portions of the body drained of blood are pale. **The intensity of the colour depends upon the**

amount of reduced haemoglobin in the blood. The colour of the hypostasis may vary from area to area in the same body.

Development: Postmortem lividity begins shortly after death, but it may not be visible for about half to one hour after death in normal individuals, and from about one to four hours in anaemic persons. **Dull-red patches of 1 to 2 cm. diameter appear in 20 to 30 minutes to two hours. These patches then deepen, increase in intensity and become confluent in one to 4 hours to reach a maximum extent and intensity within about 6 to 12 hours (Table 7-1). In the early stage, they have the same colour as blood, resembling bruises.** The patchy areas then enlarge and unite to produce extensive discolouration. Frost erythema, hypothermia-induced red-purple spots, seen over prominent parts of the body, such as shoulder, knee or elbow joints may sometimes be mistaken for hypostatic patches. **When lividity first develops, if the end of the finger is firmly pressed against the skin for a second or two, the lividity at that part will disappear and the skin will be pale or white. When the pressure is released, the lividity will reappear.** In the early stages mottled patches of hypostasis may be seen on the upper surfaces of the body, especially the legs due to uneven dilatation of the vascular bed. These patches soon join together and slide down to the dependent parts. **It is usually well developed within four hours and persists until putrefaction sets in (primary lividity).** It is present in all bodies and is more marked in bodies of fair people than in those of dark.

Intensity: It is intense in asphyxia, where the blood may not readily coagulate, and is less marked in death from wasting diseases, haemorrhage and from anaemia, due to reduced amount of blood and pigment. It is also less marked, in death from lobar pneumonia, and other conditions in which the blood coagulates quickly. **Sometimes, bluish hypostasis becomes pink along the upper part of the horizontal margin, the lower parts remaining dark. This is due to the haemoglobin being oxygenated where the erythrocytes are less densely packed in the upper layers of hypostasis.**

Table (7-1.) Hypostasis related to the time of death (hpm)

Stage	Lower limit	Upper limit
Beginning	**0.25**	**3**
Confluence	**1.0**	**4**
Maximum	**3.0**	**16**
Thumb pressure	**1.0**	**20**
Complete shifting	**2.0**	**6**
Incomplete shifting	**4.0**	**24**

Computed by Mallach from literature data.

Shifting of lividity: If the body is moved within few hours after death, patches of lividity will disappear and new ones will form on dependent parts (**secondary lividity**), but lividity to a slight degree remains in the original area, due to staining of the tissues by haemolysis (**complete shifting**). In **incomplete shifting**, after turning the body over, lividity appears slightly in the downward facing parts. **Non-displacement and non-shifting of lividity is due to haemoconcentration by loss of fluid which** penetrates the wall of those vessels related to the hydrostatic pressure.

Hypostatic congestion resembling postmortem hypostasis may be seen a few hours before death in case of a person dying slowly with circulatory failure, e.g. cholera, typhus, tuberculosis, uraemia, congestive cardiac failure, morphine poisoning and asphyxia (**Table 7-2**).

Distribution: The distribution of the stains depend on the position of the body. (1) In a body lying on its back (supine) it first appears in the neck and then spreads over the entire back extending up the flanks and sides of the neck, with the exception of the parts directly pressed on, i.e., occipital scalp, shoulder-blades, buttocks, posterior aspects of thighs, calves and heels (**Plate 2-9**). **Any pressure prevents the capillaries from filling, such as the collar band, waist band, belts, wrinkles in the clothes, etc.,** and such areas remain free from colour and are seen as strips or bands called **vibices**. This is also caused by pressure of one area of the body with another, in which case "mirror image" blanching may be seen (**contact pallor**). Such pale areas should not be mistaken for marks due to beating or when they are present on the neck, due to strangling. Hypostasis is usually well-marked in the lobes of the ears and in the tissues under nails of the fingers. **In the supine body, there may be incomplete emptying of superficial veins**

because of functioning valves in the veins draining the head. This leaves small irregular pockets of hypostasis, which may resemble contusions, particularly when putrefaction occurs. This should not be mistaken for smothering unless corroborating evidence, such as bruising of the lips or gums are present. (2) If the body is lying in prone position, the lividity appears in the loose connective tissues in front, the colour is intense and Tardieu spots are common. **Sometimes, the congestion is so great that minute blood vessels are ruptured in the nose, and cause bleeding.** This may give rise to suspicion of suffocation or strangulation. In persons who die in prone position, petechiae, ecchymoses and cutaneous blood blisters may develop after death in areas of deep hypostasis, especially in the shoulders or over the chest, which may be mistaken for asphyxial death. (3) If the body has been lying on one side, the blood will settle on that side. (4) **If the body is inverted as in drunken persons who slide out of bed, hypostasis will appear in the head and neck.** The eyes may suffuse, and many haemorrhages may appear in the conjunctivae and hypostatic areas. (5) If the body has been suspended in the upright position as in hanging, hypostasis will be most marked in the legs, external genitalia, lower parts of the forearms, and hands, and if suspension is prolonged for a few hours, petechial haemorrhages are seen in the skin. (6) In drowning, postmortem staining is usually found on the face, the upper part of chest, hands, lower arms, feet and the calves, as they are the dependent parts. If the body is constantly moving its position, as after drowning in moving water, the staining may not develop.

Fixation: Until recently it was thought that **fixation of hypostasis** was due to clotting of blood in blood vessels, but it is not correct. **The physical factors for fixation of postmortem staining are:** (1) Blood cannot pass out of the capillaries after formation of postmortem staining. (2) Rigor mortis obliterates the big vessels, and as such the blood cannot pass through these vessels to settle in venules and capillaries in a new area. (3) After full development of rigor mortis, venules and capillaries are compressed and almost empty and cannot be easily distended by the resettling blood. **Hypostasis becomes fixed when blood leaks into the surrounding soft tissues due to haemolysis and breakdown of blood vessels. This usually occurs in 6 to 12 hours.** Fixation occurs earlier in summer and is delayed in asphyxial deaths and in intracranial lesions.

Tiny haemorrhages: Tiny, often spot-like, sometimes confluent oval to round bluish-black haemorrhages (death spots, postmortem ecchymoses) exclusively limited to areas of hypostasis, result from mechanical rupture of subcutaneous capillaries and venules. They are seen commonly in the back of the shoulders and neck, and sometimes on the front of the chest, even when the body is lying on its back. **They are common in congestive types of death, may blacken the face and skin**. They are more prominent when the body lies with the head downwards.

Absence of hypostasis: Hypostasis may be sparse or even absent in deaths where blood loss of at least 65% of the circulating blood volume in adults and 45% in infant occurs before death due to trauma. In severe anaemia lividity will be absent.

Differential diagnosis: Hypostases may resemble bruises. **In some bodies, isolated patches of lividity remain separate from the large areas of lividity resembling contusions.** In doubtful cases, a portion should be removed for microscopic examination (**Plate 2-10**). As putrefaction advances, it becomes difficult to distinguish between a contusion and hypostasis.

Table (7-2). Difference between P.M. hypostasis and congestion

Trait	Hypostasis	Congestion
(1) Redness:	**Irregular and occurs on a dependent part.**	**Uniform, all over the organ.**
(2) Mucous membranes:	**Dull and lustreless.**	**Normal.**
(3) Exudate:	**No inflammatory exudate.**	**Exudate may be seen.**
(4) Hollow viscus:	**Stomach and intestine when stretched show alternate stained and unstained areas according to the position of coils.**	**Uniform staining.**

Internal Hypostasis: Hypostasis also occurs in the internal organs and the dependent parts of the liver, spleen, kidneys, intestines, larynx, lungs, heart and brain, etc. show colour changes similar to that in the skin (**Table 7-2**). Hypostasis in the heart can simulate myocardial infarction, and in the lungs it may suggest pneumonia; dependent coils of intestine appear strangulated.

Colour changes: In asphyxia the colour of the stains is deeply bluish-violet or purple. A bright pink colour is seen in hypothermia, and bodies taken from cold water as the wet skin allows atmospheric oxygen to pass through and also at low temperatures haemoglobin has a greater affinity for oxygen. This may be most marked over large joints and dependent areas. Rarely a bronze colour is seen in Clostridium perfringens septicaemias, usually associated with septic abortion. In septic abortion by Cl. welchii grayish-brown colour is seen. In mummification, lividity may turn from brown to black with drying of the body.

Colour changes in poisoning: (1) In CO poisoning the colour is cherry-red. In CO_2 poisoning, the colour is deep-blue. (2) In HCN poisoning and sometimes in burns, the colour is bright-red (**Plate 2-11**). (3) In poisoning by nitrites, potassium chlorate, potassium bicarbonate, nitrobenzene and aniline (causing methaemoglobinaemia), the colour is red-brown or chocolate-brown (**Plate 2-12**). (4) In poisoning by phosphorus, the colour is dark-brown. In poisoning by hydrogen sulphide, the colour is bluish-green.

Colour changes in decomposition: In early stages, there is haemolysis of blood and diffusion of blood pigment into the surrounding tissues where it may undergo secondary changes, e.g., sulphhaemoglobin formation. There is diffusion of blood-stained fluid in the chest or abdominal cavities. **As decomposition progresses, the lividity becomes dusky in colour and turns brown, green and black before finally disappearing with destruction of the blood.**

Effects of keeping Cadavers in Refrigerating Chamber: If the body is refrigerated soon after death, the onset of rigor mortis is delayed. Reddish patches appear on the surface, especially in the hypostatic region and sometimes in internal organs. **The blood is bright-red and injuries like abrasions and contusions have an intensified appearance.** The tissues become hard.

Medicolegal Importance: (1) It is a sign of death. (2) Its extent helps in estimating the time of death, which is not reliable. (3) It indicates the posture of the body at the time of death which is unreliable. (4) It may indicate the moving of the body to another position some time after death. (5) Sometimes, the colour may indicate the cause of death.

COLOUR MARKINGS ON DEAD BODY: Various types of colour markings may be seen on the skin or in the internal organs of dead body.

(A) Antemortem origin: (1) Trauma: Bruises and traumatic asphyxia. (2) Asphyxia: cyanosis. (3) Inflammation: congestion in organs or the skin. (4) Emboli: Fat embolism. (5) Physical: Exposure to severe cold and heat. (B) Postmortem origin: (1) Hypostasis. (2) Putrefaction. (3) Poisoning: CO, HCN, nitrites, chlorate, etc. (C) Artificial: Paint, grease, dust, mud, coal, blood, semen, etc.

MUSCULAR CHANGES

After death, the muscles of the body pass through three stages: (1) Primary relaxation or flaccidity. (2) Rigor mortis or cadaveric rigidity. (3) Secondary flaccidity.

Primary Flaccidity: During this stage, death is only somatic and it lasts for one to two hours. All the muscles of the body begin to relax soon after death. The lower jaw falls, eyelids loose tension, and joints are flexible. Body flattens over areas which are in contact with the surface on which it rests (**contact flattening**). Muscular irritability and response to mechanical or electrical stimuli persist. Peristalsis may occur in the bowel, and ciliary movements and movement of white cells may continue. Anaerobic chemical processes may continue in the tissue cells, e.g., the liver cells may dehydrogenate ethyl alcohol to acetic acid and complex chemical changes may occur in the muscles. Pupils react to atropine or physostigmine but not to light. Muscle protoplasm is slightly alkaline.

TENDON REACTION OR ZASKO'S PHENOMENON: Striking the lower third of the quadriceps femoris muscle about 10 cm. above the patella with a reflex hammer causes an upward movement of the patella because of contraction of the whole muscle. This can be seen up to 1 to 2 hours after death.

RIGOR MORTIS

Rigor mortis (death stiffening; cadaveric rigidity) is a state of stiffening of muscles, sometimes with slight shortening of the fibres. Individual cell death takes place in this stage.

Mechanism: A voluntary muscle consists of bundles of long fibres. Each fibre is formed of densely packed myofibrils extending through its whole length. These myofibrils are the contractile elements and are made up of protein filaments of two types, actin filaments and myosin filaments. In the relaxed condition, the actin filaments interdigitate with the myosin filaments only to a small extent. Under the influence of the nerve impulse, the arrays of actin filaments are drawn into the arrays of myosin filaments, rather like pistons into cylinders. This causes the muscle to contract.

During life, the separation of the actin and myosin filaments, and the energy needed for contraction are dependent on adenosinetriphosphate (ATP). The dephosphorylation of ATP by the action of ATPase produces ADP and phosphate, and a large amount of energy which is used for muscle contraction. The free phosphate then engages in a phosphorylation reaction that converts glycogen to lactic acid. The lactic acid enters blood stream and reconverted to glycogen in the liver. The lost ATP is replaced during life by resynthesis, which is dependent upon the supply of glycogen. At the time of somatic death, enough ATP is present in the muscle to maintain relaxation. **After death, the ATP is progressively and irreversibly destroyed leading to increased accumulation of lactates and phosphates in the muscles. There is no resynthesis of ATP.** The postmortem alteration of ATP is due to dephosphorylation and deamination. **Postmortem loss of integrity of the muscle cell sarcoplasmic reticulum allows calcium ions to flood the contractile units of the muscle fibres (sarcomeres) initiating the binding of actin and myosin molecules and mimicking the normal contraction process.** Normal relaxation in life is achieved by energy-dependent (ATP-driven) pumping of calcium back across the membrane of the sarcoplasmic reticulum, but this fails after death because of membrane disruption and lack of ATP, due to which **increased calcium level in the sarcomeres causes muscle contraction. When the ATP is reduced to a critical level (85% of the normal), the overlapping portions of myosin and actin filaments combine as rigid links of actomyosin, which is sticky and inextensible, and causes hardness and rigidity of muscle rigor.** The actin-myosin complex is trapped in a state of contraction until it is physically disrupted by the onset of putrefaction. This process is characterized by proteolytic detachment of actin molecules from the ends of the sarcomeres and consequent loss of the structural integrity of the contractile units. The muscles then soften and relax.

The Order of Appearance of Rigor: All muscles of the body, both voluntary and involuntary are affected. It first appears in involuntary muscles; the myocardium becomes rigid in an hour. It begins in the eyelids, lower jaw, and neck, and passes upwards to the muscles of the face and downwards to the muscles of the chest, upper limbs, abdomen, lower limbs and lastly in the fingers and toes. The progress of rigor mortis is proximo-distal. Such a sequence is not constant, symmetrical or regular. In individual limbs, it usually progresses from above downwards. **Usually it passes off in the same order in which it has appeared.** Rigor mortis always sets in, increases and decreases gradually.

Development: The development of rigor is concerned with muscles only. It is independent of the integrity of the nervous system, though it is said to develop more slowly in paralysed limbs. Before rigor mortis develops, the body can be moved to any posture, and the rigor will fix in that posture. **When rigor is developing the extremities can be moved and the rigor, temporarily overcome, develops later and fixes the extremities in their new position,** but the rigidity will be less than other symmetrical groups, which have been not disturbed. **If force is applied when rigor is fully developed, stiffness is broken up permanently** and the rigid muscles may show postmortem ruptures. Frequent handling of the body breaks the rigor in certain places.

Features: When rigor is fully developed, the entire body is stiff, the muscles shortened, hard and opaque; knees, hips, shoulders and elbows are slightly flexed and fingers and toes often show a marked degree of flexion. Rigor of erector pilae muscles may cause roughness of the skin known

as **cutis anserina** or goose skin. The testes may be drawn up into the groin; semen may be forced out of the seminal vesicles, and the pupils may be partially contracted. Postmortem emission of semen may occur agonally or later, due to rigor mortis in the dartos of the scrotum, and has no significance. Rarely, if the uterus is in labour at the time of death, the rigor mortis may cause the uterus to contract and expel the foetus.

Testing: Rigor is tested by trying to lift the eyelids, depressing the jaw and gently bending the neck and various joints of the body. Note the degree (absent, minimal, moderate, advanced or complete) and distribution.

Time of Onset: In India, it begins one to two hours after death and takes further one to two hours to develop and gets well established in the entire-body in six hours in summer. In temperate countries, it begins in three to six hours and takes further two to three hours to develop. Rule of 12 (March of rigor) is not applicable in tropical countries.

Duration of Rigor Mortis: In India, usually it lasts 24 to 48 hours in winter and 18 to 36 hours in summer. It may begin to disappear in about 12 hours in summer. These times are variable because of many intrinsic and extrinsic factors. It lasts for two to three days in temperate regions. When rigor sets in early, it passes off quickly and vice versa.

Conditions Altering the Onset and Duration:

(1) AGE: Rigor does not occur in a foetus of less than seven months. In healthy adults, it develops slowly but is well-marked, while in children and old people it is feeble and rapid.

(2) NATURE OF DEATH: The onset of rigor is early and duration is short in deaths from diseases causing great exhaustion and wasting, e.g., cholera, typhoid, tuberculosis, cancer, etc., and in violent death as by cut-throat, firearms, electrocution, lightning and strychnine poisoning. The onset is delayed in deaths from asphyxia, severe haemorrhage, apoplexy, pneumonia, and nervous disease causing paralysis of muscles. In deaths due to poisoning from HCN and strychnine, it starts early and persists longer.

(3) MUSCULAR STATE: The onset is slow and the duration long, in cases where muscles are healthy and at rest before death. The onset is rapid if there is fatigue or exhaustion before death.

(4) ATMOSPHERIC CONDITIONS: The onset is slow and duration long in cold weather. The onset is rapid and duration short due to heat, because of the increased breakdown of ATP. It may persist for three to four days in refrigerated conditions.

Because of the number and variability of the factors which influence the development of rigor mortis, **it is not possible to deduce any general rule for the rate of its onset, duration and disappearance.**

Medicolegal Importance: (1) It is a sign of death. (2) Its extent helps in estimating the time of death, which is not reliable. (3) It indicates the position of the body at the time of death.

Conditions Simulating Rigor Mortis

(1) Heat Stiffening: When a body is exposed to temperatures above 65°C., rigidity is produced which is much more marked than that found in rigor mortis. It is seen in deaths from burning, high voltage electric shock and from falling into hot liquid. Heat causes stiffening of the muscles, because the tissue proteins are denatured and coagulated as in cooking. The muscles are contracted, dry, or even carbonized on the surface. Changes in posture, especially flexion of the limbs occurs due to muscle contraction. Heat stiffening cannot be broken down by extending the limbs as in rigor mortis. **The stiffening remains until the muscles and ligaments soften from decomposition, and the normal rigor mortis does not occur.**

(2) Cold Stiffening: When a body is exposed to freezing temperatures, before acid metabolites appear in the muscles, the tissues become frozen and stiff, due to freezing of the body fluids and solidification of subcutaneous fat, simulating rigor. The body is extremely cold and markedly rigid. When the joints are forcibly flexed, ice breaks in the synovial fluid with a sudden sharp sound. **If the body is placed in warm atmosphere, the stiffness disappears and after a time, the normal rigor mortis occurs.**

(3) CADAVERIC SPASM OR INSTANTANEOUS RIGOR

Mechanism: The mechanism of cadaveric spasm is obscure, but possibly may be neurogenic and not the same chemical process as true rigor. The failure of chemical processes required for active muscular relaxation, or adrenocortical exhaustion which impairs resynthesis of ATP may be the possible causes.

Muscles involved: Cadaveric spasm (cataleptic rigidity) is a rare condition. **In this, the group of**

muscles which have been actively working and contracted at the moment of death, become stiff and rigid immediately after death, without passing into the stage of primary relaxation. As such, the change preserves the exact attitude of the person at the time of death for several hours afterwards (**Fig. 7-2**). The spasm is primarily a vital phenomenon as it originates by normal nervous stimulation of the muscles. This is usually limited to a single group of voluntary muscles and frequently involves the hands; occasionally the whole body is affected, as seen in soldiers shot in battle when the body may retain the position and attitude which it assumed at the moment of death (**Table 7-3**).

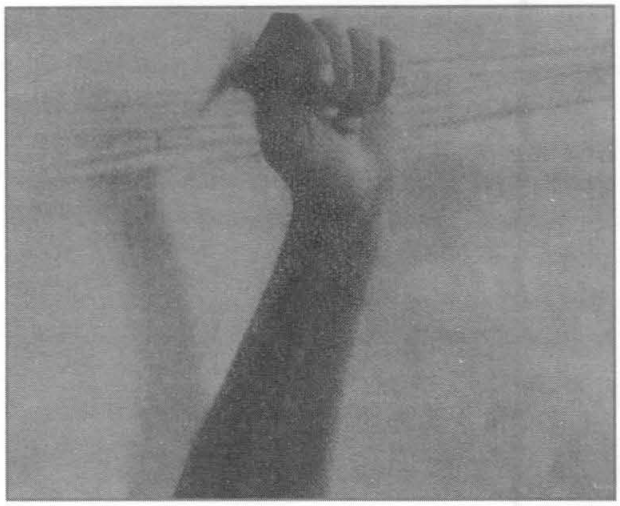


Fig. (7-2). Cadaveric spasm. The indoor aerial is seen firmly grasped in the hand causing electrocution.

Predisposing factors: It occurs especially in cases of intense physical and/or emotional activity, such as excitement, fear, severe pain, exhaustion, cerebral haemorrhage, injury to the nervous system, firearm wound of the head, drowning, convulsant poison, such as strychnine, etc.

No other condition simulates cadaveric spasm, and it cannot be produced by any method after death. Very great force is required to overcome stiffness. It passes without interruption into normal rigor mortis, and passes off when rigor disappears.

Medicolegal importance: (1) Occasionally, in case of suicide the weapon, e.g., pistol or knife is seen firmly grasped in the victim's hand, which is a strong presumptive evidence of suicide (**Fig. 7-2**). Attempts may be made to simulate this condition in order to mask murder. But, ordinary rigor does not produce the same firm grip of a weapon, and the weapon may be placed in the hand in a way which could not have been used by a suicide. (2) If the deceased dies due to assault, some part of clothing, e.g., button of his assailant or some hair may be firmly grasped in the hands. (3) In case of drowning, material such as grass, weeds or gravel may be found firmly grasped in the hands, which indicates that the victim was alive on entering the water (**Table 7-3**).

Table (7-3). Difference between rigor mortis and cadaveric spasm

	Trait	Rigor mortis	Cadaveric spasm
(1)	**Production:**	**Freezing and exposure to temperature above 65° will produce rigor.**	**Cannot be produced by any method after death.**
(2)	**Mechanism:**	**Known. Reduction of ATP.**	**Not clearly known.**
(3)	**Predisposing factors:**	**Nil.**	**Sudden death, excitement, fear, exhaustion, nervous tension, etc.**
(4)	**Time of onset:**	**One to two hours after death.**	**Instantaneous.**
(5)	**Muscles involved:**	**All the muscles of the body, both voluntary and involuntary.**	**Usually a single group of voluntary muscles.**
(6)	**Muscle stiffening:**	**Not marked: moderate force can overcome it.**	**Marked: great force is required to overcome it.**
(7)	**Molecular death:**	**Occurs.**	**Does not occur.**
(8)	**Body heat:**	**Cold.**	**Warm.**
(9)	**Electrical stimuli:**	**Muscles do not respond.**	**Muscles respond.**
(10)	**Muscular reaction:**	**Acidic.**	**Alkaline.**
(11)	**Medicolegal importance:**	**Indicates time of death.**	**Indicates mode of death, i.e., suicide, homicide, or accident.**

Secondary Relaxation: Muscles become soft and flaccid due to the action of the alkaline liquids produced by putrefaction. Another view is that rigidity disappears due to solution of myosin by excess of acid produced during rigor mortis. A third view is that enzymes are developed in dead muscles which dissolve myosin by a process of autodigestion.

DECOMPOSITION

It involves two processes: (1) Autolysis. (2) Putrefaction.

Autolysis: Autolysis is self-digestion of tissues. Soon after death, cell membranes become permeable and breakdown, with release of cytoplasm containing enzymes. Lysosomes and their digestive enzymes (mainly hydrolases) are released from the cells. The proteolytic, glycolytic and lipolytic action of ferments causes autodigestion and disintegration of organs without bacterial influence. This chemical process is increased by heat and is stopped by freezing or inactivation of enzymes by the heat. The earliest autolytic changes occur in parenchymatous and glandular tissues and in the brain. The lining of intestines, adrenal medulla and pancreas autolyse within hours of death. Autolytic fermentation results in maceration of the dead foetus in uterus, early softening and liquefaction of the brain of the newborn and infants, and softening of the internal organs. The earliest external sign is a whitish, cloudy appearance in the cornea.

PUTREFACTION: The terms decomposition and putrefaction are used as synonyms. It is the final stage following death.

Mechanism: Destruction of the soft tissues of the body is caused, mainly by the action of bacterial enzymes, mostly anaerobic organisms derived from the intestines. Other enzymes are derived from fungi, such as Penicillium and Aspergillus, and sometimes from insects. **The chief destructive bacterial agent is Cl. welchii, which causes marked haemolysis, liquefaction of postmortem clots and of fresh thrombi and emboli, disintegration of tissues and gas formation in blood vessels and tissue spaces. Lecithinase produced by Cl. welchii is most important.** This hydrolyse the lecithin which is present in all cell membranes including blood cells, and is responsible for the postmortem haemolysis of blood. The other organisms include streptococci, staphylococci, B. proteus, B. coli, B. aerogenes capsulatus, bacteroides, anaerobic lactobacilli, diphtheroids, etc. Bacteria produce a large variety of enzymes and these breakdown the various tissues of the body. It begins immediately after death at the cellular level. There is progressive breakdown of soft tissues and the alteration of their proteins, carbohydrates and fats. **Organisms enter the tissues shortly after death mainly from the intestines, and less often through the respiratory tract, or through external skin wounds.** The fall in the oxygen concentration in the tissues and rise in hydrogen ion concentration after death, favour bacterial growth and spread throughout the body. Because the protective agencies of the body are absent, the bacteria spread through the blood vessels, using the proteins and carbohydrates of the blood as culture media.

Features: The characteristic features of putrefaction are: (1) changes in the colour of the tissues, (2) collection of gases in the tissues, and (3) liquefaction of tissues. The same changes seen on the surface of the body occur simultaneously in internal organs. **The exact chronological order of the appearance of putrefactive changes is highly variable**, and depends on a broad variety of individual as well as environmental conditions. As decomposition sets in, joints become lax and loss of tension in intervertebral discs tends to shorten the spinal column and total height by one centimetre.

(1) Colour Changes: External: Bacteria spread directly from the bowel into the tissues of the abdominal wall. They spread most easily in fluid and tend to colonise the venous system. The **first external sign of putrefaction in a body lying in air is usually a greenish discolouration of the skin over the region of the caecum, where the contents of the bowel are more fluid and full of bacteria** (Plate 3-13). **Internally, this is seen on the undersurface of the liver where that organ is in contact with the transverse colon.** Greenish colouration is due to the conversion of haemoglobin into sulphmethaemoglobin, by the hydrogen sulphide formed in the large intestine and escaping into the surrounding tissues. **The colour appears in 12 to 18 hours in summer and in one to two days in winter.** Green colouration is more easily seen on a fair skin than on a dark one. **The green colouration then spreads over the entire abdomen, external genitals, and then patches appear successively on the chest,**

Fig. (7-3). Putrefactive network (marbling).

neck, face, arms and legs. The patches become dark-green and later purple and dark-blue. They are at first scattered, but later on unite and the whole skin of the body appears discoloured.

Marbling of skin: The superficial veins, especially over the roots of the limbs, shoulders, chest, sides of the abdomen and neck are stained greenish-brown or purplish-red depending on the total amount of sulph-haemoglobin formation within the affected vessels, due to the haemolysis of red cells which stains the wall of the vessel and infiltrates into the tissue, giving a **marbled** appearance (red, then greenish pattern in skin resembling the branches of a tree) (**Plate 3-15**). **This starts in 24 hours but is prominent in 36 to 48 hours.** Marbling is often first seen in the vessels close to the caecum and sigmoid colon (**Fig. 7-3**).

Fluidity of blood: The clotted blood becomes fluid, and as such, the position of the postmortem staining is altered and the fluid blood collects in the serous cavities. Putrefactive effusion of bloodstained fluid into the pleural cavities usually starts at about the time when the skin becomes macerated. Such effusions usually do not exceed 60 to 100 ml. unless death resulted from drowning. In advanced putrefaction, conjunctival petechiae may not be distinguished due to haemolysis, and the conjunctivae have a homogeneous grayish to light-greenish appearance.

Internal: The earliest internal change is a reddish-brown discolouration of the inner surface of the vessels, especially of the aorta. Internally, the same changes of colour are seen in the viscera, but the colour varies from dark-red to black, rather than green and produces the appearance of congestion. With this colour change, the viscera become softer and greasy to touch. Finally, they breakdown into soft disintegrating mass.

(2) Development of Foul-smelling Gases: The chemical processes in this stage are those of reduction, the **complicated proteins and carbohydrates being split into simpler compounds.** The gases are non-inflammable in the early stages, but as the decomposition progresses, enough of hydrogen sulphide is formed, which can be ignited to burn with a blue flame. **Gases collect in the intestines in 12 to 18 hours in summer and in one to 2 days in winter,** and the abdomen becomes tense and distended. At about the same time, the eyeballs become soft and yielding, the cornea becomes white and flattened. Later, the eyes collapse.

Gas bubbles accumulate in the tissues, causing crepitant, sponge-like feeling which soon begins to distend the body. **From 18 to 36 hours after death, the gases collect in the tissues, cavities and hollow viscera under considerable pressure, and the features become bloated and distorted.** The gas formation in the blood vessels may force bloodstained fluid, air or liquid fat between the epidermis and dermis forming **small blisters in 18 to 24 hours (Fig. 7-4).** Blisters are formed first on the lower surfaces of trunk and thighs, where tissues contain more fluid due to hypostatic oedema. These gradually enlarge, unite and rupture, exposing large areas of dermis. On opening the abdomen the gas escapes with a loud explosive noise. **Discoloured natural fluids and liquefied tissues are made frothy by gas. Gases in the abdomen,**

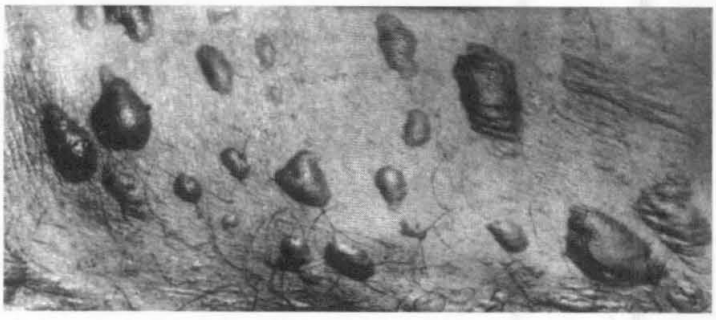


Fig. (7-4). Blisters of putrefaction.

force the diaphragm upwards compressing the lungs and heart, and bloodstained froth exudes from the mouth and nostrils (postmortem purge), which can be mistaken for bleeding following antemortem injury. The compression of heart forces out its contents. Pressure of the gases may force food from the stomach into the fauces, and this may fall into the larynx. **In 24 to 48 hours, the subcutaneous tissues become emphysematous, due to which even a thin body appears obese.** The breasts, and penis are greatly distended. In males, gas is forced from the peritoneal space down the inguinal canal and up to the scrotum, resulting in massive scrotal swelling. The eyes bulge from their sockets, the tongue is forced out between the swollen and discoloured lips **(Fig. 7-5 and Plate 3-14)**. The sphincters relax and urine and faeces may escape. **The maggots are produced in one to 2 days which have proteolytic enzymes and penetrate the skin fairly rapidly (Plate 3-16)**. This allows air to enter under the skin and more maggots into the body cavities. The activities of maggots may raise the temperature to near or even above that of normal body heat. The anus and uterus may prolapse after two to three days, and postmortem delivery of a foetus may take place. Wounds caused before or after death bleed, due to pressure of the gases within the heart and blood vessels, and whether they are antemortem or postmortem cannot be made out. **After three days, the face is so discoloured and bloated that identification becomes very difficult (Fig. 7-5).** Blood stained fluid (tissue liquefaction stained by haemolysis) may leak from mouth, nostrils, rectum and vagina. The hair becomes loose and is easily pulled out. The nails are also loose. In three **to five days or more, the sutures of skull especially of children are separated and liquid brain comes out.** Teeth become loose and may fall out. **The skin shows "slippage", and the skin of the hands and feet may come off in a "glove and stocking" fashion.** When the nutrient material is used up, the formation of gas stops and the swelling gradually subsides. The gas leaves the tissues, usually by escaping as a result of damage to the structures or by drainage through a postmortem wound.

(3) Liquefaction of Tissues: Colliquative putrefaction begins from five days to ten days or more after death. In fat people, the fat, especially

Fig. (7-5). Advanced decomposition showing marked gaseous swelling of face, eye balls and tongue.

omental and mesenteric may liquefy into a translucent yellow fluid. **The abdomen bursts and the stomach and intestines protrude.** In children, thorax also bursts. **The tissues become soft, loose and are converted into a thick, semifluid, black mass and are separated from the bones and fall off.** The cartilages and ligaments are softened in the final stage. The body lying in air decomposes usually in the following order: abdomen, chest, neck, face, legs, shoulders and arms.

Rigidity due to Inflation of the Tissues with Gases: In this condition, changes of decomposition are well-marked. **False rigidity is produced due to accumulation of gases in the tissues because of rapid decomposition of tissues that have imbibed much fluid.** The lower limbs are abducted, flexed, and rigid, the arms are abducted and flexed, the hands are open and the fingers are wide apart. The rigidity persists till the escape of gases due to advancing decomposition. **This condition is especially seen in bodies recovered from water.**

Differential decomposition: Decomposition may differ from body to body, from environment to environment and from one part of the same body to another. It means a situation where one body shows differential stages of decomposition in different parts. Sometimes, one part of the body may be mummified, while the rest may show liquefying putrefaction.

INTERNAL PHENOMENON: As the blood decomposes, haemoglobin transudes into the tissues, which gradually change to greenish-yellow, greenish-blue, greenish-black and finally black. The viscera become greasy and softened. As a general rule, the organs show putrefactive changes in the following order: (1) Larynx and trachea. (2) Stomach, intestines and spleen. (3) Liver and lungs. (4) Brain. (5) Heart. (6) Kidney and bladder. (7) Prostate and uterus. (8) Skin, muscle, tendon. (9) Bones.

SPLEEN: It becomes soft, pulpy and liquefies in two to three days.

LIVER: It becomes soft in 12 to 24 hours in summer and studded with blisters in 24 to 36 hours. Cl. welchii form characteristic small clumps in a tissue space and produce gas, which soon increase in size. These lesions appear first as small opaque, yellowish-gray, dendritic figures in the parenchyma. When bubbles develop, the organ has a honeycombed, vesicular or "foamy" appearance (Fig. 7-6). The greenish discolouration extends to the whole organ, which finally becomes coal-black.

BRAIN: In summer, it becomes soft and pulpy in 1 to 2 days, and liquefies in 3 to 4 days.

HEART: It becomes soft and flabby and the cavity appears dilated.

LUNGS: Gaseous bullae are formed under the pleura which are small, pale-red and scattered, and later combine. Later the lungs become soft, collapse and are reduced to a small black mass.

KIDNEYS: The renal tissue becomes flabby and the surfaces are dull and appear par-boiled.

PROSTATE: It resists putrefaction for a long time.

UTERUS: The virgin uterus is the last organ to putrefy. Gravid uterus or soon after delivery, it rapidly putrefies.

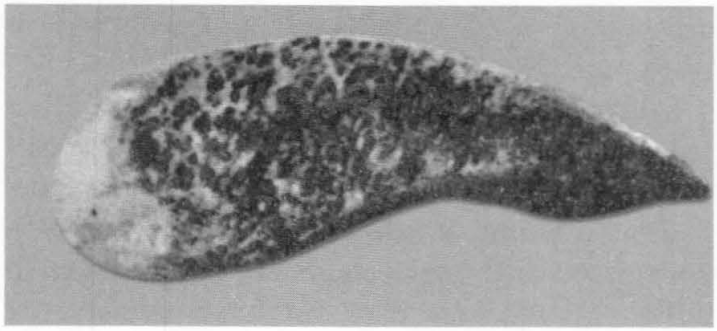


Fig. (7-6). Cross-section of liver showing honeycomb appearance due to putrefaction.

CONDITIONS AFFECTING THE RATE OF PUTREFACTION

(A) EXTERNAL: (1) TEMPERATURE: Putrefaction begins above 10°C and is optimum between 21°C and 38°C. It is arrested below 0°C and above 48°C. The rate of decomposition is about twice as rapid in summer as in winter. Advanced putrefaction may be seen within 24 to 36 hours in summer. A frozen body will not undergo decomposition until it defrosts.

(2) MOISTURE: For putrefaction moisture is necessary, and rapid drying of the body practically inhibits it. After death in generalised oedema, putrefaction is very rapid, and bodies recovered from water, if left in the air decompose rapidly. Organs which contain water decompose more readily.

(3) AIR: Free access of air hastens putrefaction. Moist and still air helps putrefaction.

(4) CLOTHING: Initially, clothing hastens putrefaction by maintaining body temperature. If the clothing is tight as under belts, socks, boots, and tight-fitting undergarments, the putrefaction is slow for it causes compression of the tissues, which drives out the blood from the part, and prevents the entry of internal organisms.

(5) MANNER OF BURIAL: Putrefaction is rapid in a body buried in a damp, marshy or shallow grave without clothes or coffin, because the body is exposed to constant changes of temperature. In acid peaty soils even the bones may be destroyed. Putrefaction is delayed if body is buried in dry, sandy soil, or in a grave deeper than 2 metres and when the body is covered and placed in a coffin. When a body is buried in lime, decomposition is delayed.

(B) INTERNAL: (1) AGE: The bodies of newborn children who have not been fed decompose very slowly, because the bodies are normally sterile. Bodies of children putrefy rapidly and of old people slowly.

(2) SEX: Sex has no effect.

(3) CONDITION OF THE BODY: Fat bodies putrefy quickly than lean bodies, due to larger amount of fluid in the tissues and excess fat, and greater retention of heat.

(4) CAUSE OF DEATH: Bodies of persons dying from septicaemia, peritonitis, inflammatory and septic conditions, general anasarca, asphyxia, etc., decompose rapidly. Putrefaction develops very rapidly in infection due to Cl. welchii, e.g., acute intestinal obstruction, some cases of abortion and gas gangrene. Putrefaction is delayed after death due to wasting disease, anaemia, debility.

(5) MUTILATION: Bodies in which there are wounds, or which have suffered from other forms of violence before death putrefy rapidly, because the organisms can easily enter damaged tissues. In case of dismemberment, the limbs putrefy slowly, because they are drained of the blood, and intestinal organisms do not gain entry. The trunk putrefies rapidly because of the action of intestinal bacteria and the airborne organisms.

BODY FARM: It is a research facility dedicated to the estimation of time of death. Bodies are exposed to different ways to decompose. In all stages of decay, animal activity, smell, body temperature and weather conditions are recorded, for gaining a better understanding of decomposition process.

Putrefaction in Water: Casper dictum states that a body decomposes in air twice as rapidly as in water, and eight times as rapidly as in earth.

The variations are very great, and it is not of much practical value. Putrefaction is more rapid in warm, fresh, and stagnant water, than in cold, salty and running water. Putrefaction is rapid in a body lying in water contaminated with sewage, but is delayed when a body is lying in deep water and is well protected by clothing. As the submerged bodies float face down, with the head lower than the trunk, gaseous distension and postmortem discolouration are first seen on the face and then spread to the neck, upper extremities, chest, abdomen and the lower extremities in that order. The epidermis of the hands and feet becomes swollen, bleached and wrinkled after immersion and may be removed as a cast of the extremity after two to three days. After several weeks in water, macerated flesh may be stripped from the body by the action of currents or the contact with the floating objects. Fish, crustacea (crab, lobster, shrimp, etc.) and water-rats near a sewer may destroy the body.

SKELETONISATION: The time required for skeletonisation varies considerably. It mainly depends on the ambient temperature, insect colonisation of the body and scavenger activity. In a hot humid environment with heavy insect activity, a corpse can be skeletonised in three days. In the case of an exposed body, flies, maggots, ants, cockroaches, rats, dogs, jackals, vultures, etc., may reduce the body to a skeleton within a few days. When the body is in water, it may be attacked by fishes, crabs, etc., which reduce the body to a skeleton in a few days. In a deeply buried body, the lower temperature, the exclusion of air, absence of animal life, etc., delay the decomposition markedly. In bodies placed in airtight coffins, decay process may not occur for several decades. In India, an uncoffined buried body is reduced to a skeleton within about a year. Buried bones may decay at different rates, e.g. neutral soil may not destroy the skeleton at all. Acidic soil may cause decay in about 25 to 100 years. In a hot climate, bones on the ground surface may decay in 5 to 10 years. Flat bones and the bones of the infants and old breakdown faster.

ADIPOCERE (SAPONIFICATION)

It is a modification of putrefaction.

Mechanism: In this, the fatty tissues of the body change into a substance similar to soaps, known as adipocere. The change is due to the gradual hydrolysis and hydrogenation of preexisting fats, such as olein, into higher fatty acids, which being acidic, inhibit putrefactive bacteria. Ultimately, the whole of the fat is converted into palmitic, oleic, stearic and hydroxystearic acids, together with some glycerol, and a mixture of these substances forms adipocere. **The process starts under the influence of intrinsic lipases, and continued by the bacterial enzymes, especially those of the clostridia group.**

Factors influencing adipocere formation: Water is essential for the bacterial and enzymatic processes involved in adipocere formation. The water required for the hydrolysis is obtained mainly from the body tissues, which therefore become more and more dehydrated. In a body immerzed in water, this fluid contributes to the hydrolysis of the subcutaneous fat. **A warm, moist, anaerobic environment favours adipocere formation.** It is more frequently seen in females, the well-nourished, mature newborn children, in the obese and in corpses that have been submerged in water for a long period. It is also seen in buried bodies. **Foetuses under seven months do not show this change.** The bodies enclosed in a water-tight coffin for many years may be converted to adipocere even in the absence of external water. Adipocere is delayed by cold and formed rapidly by heat.

Properties: Adipocere has a distinct offensive or sweetish smell, but during the early stages of its production, a penetrating ammoniacal odour is noted. **The smell remains in the clothing of those handling such bodies for several days.** Fresh adipocere is soft, moist, whitish, translucent, and greasy, resembling pale, rancid butter. After some years it becomes dry, hard, brittle, cracked and yellowish. It is inflammable and burns with a faint-yellow flame. It floats in water and dissolves in alcohol and ether.

Distribution: It forms in any site where fatty tissue is present. It is formed first in the subcutaneous tissues. The face, buttocks, breasts and abdomen are the usual sites. The limbs, chest wall, or other parts of the body may be affected. In most cases, it is partial and irregular. **Sometimes the entire body is converted into adipocere.** Depending upon mostly on temperature and also humidity of environment **(Plate 3-17 and 3-18)**. Fatty tissue between the fibres of skeletal muscle and in the myocardium, and in the substance of the liver, kidney, etc., is also converted into adipocere. **The epidermis disappears as adipocere forms.** Small muscles are dehydrated and become extremely thin, and have a uniform grayish colour. The intestines and lungs are usually parchment-like in consistency and thinness. **The liver is prominent and retains its shape. Histologically, the gross features of the organs can sometimes be appreciated, even though the cells are lacking.** In most cases, the formation of adipocere is partial and irregular. Adipocere may persist for decades.

Time Required for Adipocere Formation: The rate of adipocere formation is extremely variable. In temperate countries, the **shortest time for its formation is about three weeks in summer,** when it occurs to a certain extent. Complete conversion in an **adult limb requires at least three to six months.** Foetuses under seven months do not show this change. In India, it has been observed to begin within 4 to 5 days.

Medicolegal Importance: (1) When the process involves the face, the features are well preserved, which help to establish the identity. (2) The cause of death can be determined, because injuries are recognised. (3) The time since death can be estimated, which is not reliable.

MUMMIFICATION

It is a modification of putrefaction. **Dehydration or drying and shrinking of the cadaver occur from the evaporation of water, but the natural appearances of the body and general facial features are preserved.**

Features: It begins in the exposed parts of the body like face, hands and feet and then extends to the entire body including the internal organs. The skin may be shrunken and contracted, dry, brittle, leathery and rusty-brown in colour and adheres closely to the bones **(Fig. 7-7)**. As the skin contracts, some of the fat cells in the subcutaneous tissues are broken and the liquid oil is forced into the dermis which becomes translucent. **The entire body loses weight, becomes thin, stiff and brittle.** In mummified bodies, arms are often abducted in the shoulder joints, flexed in elbow joints, and hands are clenched into fists in most cases. This flexion is often seen in lower limbs also. **This is due to shrinkage of muscles and tendons.** Mummification may be partial in some cases with only limbs or head or trunk being affected. Mummified tissues are dry, leathery and brown in colour. The **internal organs are shrunken, hard, dark-brown and black and mix together or may disappear due to putrefaction and maggot activity. If a mummified body is not protected, it will be gradually reduced to skeleton, though tough, leathery shreds of skin tendons and ligaments may persist for many years,** but if protected, it may be preserved for years. A mummified body is practically odourless. The **time required for complete mummification of a body varies from three months to a year or two (Fig. 7-7)**.

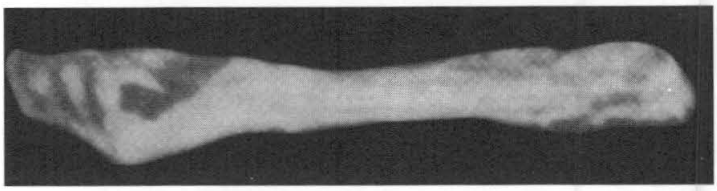


Fig. (7-7). Mummified forearm.

Factors necessary for the production of mummification: (1) **The absence of moisture in the air,** and (2) the **continuous action of dry or warmed air.** Marked dehydration before death favours the development of mummification. Mummification of newborn children may occur, if they are left in a trunk, or a kitchen cupboard where the atmosphere is warm and dry. Mummification occurs in bodies buried in shallow graves in dry sandy soils, where evaporation of body fluids is very rapid due to the hot dry winds in summer.

Occasionally, a body which shows evidence of mummification in certain parts, may show changes of adipocere in others. **(differential decomposition)**.

Medicolegal Importance: It is the same as that of adipocere.

CONDITIONS PRESERVING THE BODY

(1) Embalming: Embalming is treatment of the dead body with antiseptics and preservatives to prevent putrefaction. In this the contents of the gastrointestinal tract, the bladder and the blood vessels are removed by a trocar and suction apparatus through a puncture wound of the abdomen. Venous drainage either from the internal jugular vein or axillary vein should be done. **A typical embalming fluid consists of** formalin one-and-half litres; sodium citrate 900 g.; sodium chloride 800 g.; sodium borate 600 g.; glycerine 600 ml.; one per cent eosin 30 ml. and water up to ten litres. This is a medium hard solution containing six per cent of formalin. **In an adult, ten litres of embalming fluid is injected through the axillary, carotid or femoral artery** by: (1) gravity feed injector, (2) bulb syringe, (3) hand pump, or (4) motorised pump injector. One litre of 40% formalin containing 25% methanol and ten percent phenol should be injected distributing it evenly throughout the thoracic and abdominal cavities. By this process proteins are coagulated, tissues are fixed, organs are bleached and hardened and blood is converted into a brownish mass. **Decomposition is inhibited for many months, if the injection is made shortly after death. Embalming produces a chemical stiffening similar to rigor mortis, and normal rigor does not develop.** Embalming alters the appearance of the body, tissues and organs, making it difficult to interpret any injury or disease. **It becomes very difficult to extract drugs and determine the presence of many poisons.** Blood grouping cannot be made out. Thrombi and emboli will be dislocated and washed away.

(2) Mummification.

(3) Adipocere formation.

(4) Freezing: If the body is frozen soon after death and kept in that state, it will be preserved for decades.

(5) Bodies which have been in water or soil containing **antiseptic substances,** sometimes become impregnated with these material and do not decompose.

ESTIMATION OF POSTMORTEM INTERVAL

The interval between death, and the time of examination of a body is known as postmortem interval. This is important (1) to know when the crime was committed, (2) gives the police a starting point for their inquiries, (3) to exclude some suspects, and (4) to check on a suspect's statement.

The exact time of death cannot be fixed by any method, but only an approximate range of time of death can be given, because there are considerable biological variations in individual cases. The opinion should be based on correlation of evidence from all available sources, along with postmortem findings. **One should never give a single estimate of the time since death, but use a range of times, between which the death was presumed to have taken place. The range of time provided is at best an educated guess and subject to error.** The points to be noted are: (1) Cooling of the body. (2) Postmortem lividity. (3) Rigor mortis. (4) Progress of decomposition, adipocere and mummification.

(5) Entomology of the Cadaver: Entomology is the study of the form and behaviour of insects. Myiasis is a condition caused by infestation of the body by fly maggots. Flies (Musca domestica and M. vicina) **may deposit their eggs (pearly-white, one mm long; about 120 to 150 eggs at one sitting) between the lips or the eyelids, in the nostrils or in the margins of a fresh wound, any natural or traumatically created shaded orifices, such as hair and the ground-body interface, within a few minutes after death. In 8 to 24 hours in summer, larvae or maggots (white, segmented, 1 to 2 mm. in length at birth; 12 mm. when fully grown) are produced from the eggs which crawl into the interior of the body, and produce powerful proteolytic enzymes, and destroy the soft tissues. The maggots become pupae (dark-brown, barrel-shaped, 6 mm length) in 3 to 6 days and the pupae become adult flies in 3 to 6 days. The complete lifecycle from egg to adult may take 5 to 6 days in summer, and 8 to 20 days in winter. Body lice usually remain alive for 3 to 6 days, after the death of a person.**

(6) Gastrointestinal Tract: Emptying of Stomach: There is considerable variation in emptying of the stomach. **The gastric emptying of either liquids or solids is subject to relatively wide differences in the same and different individuals even if the same meal is ingested. The emptying time of the stomach varies from person to person, and even in the same person at different times, depending on the nature of the food.** The stomach contents do not enter the duodenum after death. The stomach empties gradually. **The bulk of the meal leaves the stomach within 2 hours. A light**

meal usually leaves the stomach within 2 hours after being eaten, a medium-sized meal requires 3 to 4 hours, and a heavy meal 5 to 8 hours. Meat, green vegetables and roots cannot be made out after 4 hours. A carbohydrate meal leaves the stomach more rapidly than a protein meal, because carbohydrates are reduced to a semi-fluid state rapidly, and a protein meal leaves the stomach more rapidly than fatty meal as fats inhibit gastric motility. The state of digestion and transportation rate of food from the stomach into the duodenum depends on several factors, such as anatomical, physiological, pathological, psychological, agonal, kind of food, etc., which contribute greatly to intra-and inter-individual variability of gastric emptying. **The quantity and digestive state of gastric contents is modified by the following factors.** (1) The total quantity of food taken at a meal. (2) Additional snacks taken between a meal. (3) The ratio of solid to liquid in the meal. (4) The carbohydrate/fat content. (5) Marked variation between individuals. (6) Variation in the same individual from day to day. (7) Dramatic variations due to psychogenic and endocrine factors.

Variability in gastric emptying: In head injury, undigested food may be seen after a period of coma of more than twenty four hours. Digestion is delayed during sleep, and is probably suspended during coma. Any illness or emotional stress, e.g., fear, worry, psychogenic pylorospasm may prolong the emptying time for many hours. Strong alcohols irritate the mucosa and tend to delay emptying. Hypermotility caused by emotional disturbance, can result in rapid passage of food through the gut causing diarrhoea.

The head of the meal reaches the hepatic flexure in about six hours, splenic flexure in nine to twelve hours, and pelvic colon in 12 to 18 hours.

(7) Cerebrospinal Fluid: Cisternal fluid is examined. The amount of potassium increases at a constant rate in relation to the temperature of the body during the first 20 hours. The concentrations of sodium, calcium and magnesium have no obvious relationship to time since death.

(8) Blood: There is a progressive increase of lactic acid. The values increase by 50 to 75 fold in 12 to 24 hours, the steepest increase occurring in first 6 to 8 hours. Amino acid nitrogen is less than 14 mg/dl up to 10 hours, but rises to 30 mg/dl by 48 hours. Acid phosphatase levels increase 20 times by 48 hours. Amylase levels increase 3 to 4 times on second day. Glutamate-oxalate transaminase (AST4) and lactate dehydrogenase (LDH) increase linearly over the first 60 hours. Sodium levels fall by 0.9 mEg/l per hour. Organic phosphorus level in serum reaches 20 mEg/l at 18 hours after death.

(9) Pericardial Fluid: 10 to 15 ml can be obtained, as opposed to 1 to 2 ml of vitreous humour and 5 ml of CSF. Constituents are not helpful in estimating time since death.

(10) Synovial Fluid: There is linear rise of potassium, which increases more than double within the first two days.

(11) Vitreous Humour: There is a linear relationship between vitreous potassium concentration and time elapsed after death up to 120 hours. The 95% limits of confidence are ±20 hours up to 100 hours. The rise is mainly due to diffusion from the retina into the center of the globe. There is a linear rise of hypoxanthine (Hx) up to 120 hours.

(12) Muscle Enzymes: Myofibrillar protease activity increases linearly and creatinine phosphokinase decreases linearly after death. A strong positive correlation is found between the ratio of non-protein nitrogen and total soluble protein and also creatinine concentration and time since death.

All the chemical methods for estimating the time since death are temperature-dependent. Chemical methods are of limited practical value. They are widely considered as the least reliable and the least practicable of all other methods.

(13) Hair: Hair does not grow after death; the contraction of the skin towards the hair roots gives the illusion of growth. A sample of hair is shaved from the chin and its length measured. From this, a rough estimation of the time since the last shave can be made, for beard hair grows at the rate of 0.4 mm. per day.

(14) The Scene of Death: The dates on mail or newspapers, sales receipts or dated slips of paper in the deceased's pockets, when the neighbours last saw the person, degree of coagulation of milk, state of food on a table, etc. may be valuable. The state of dress should be noted as regards whether the person is fully dressed or in the night dress. If the watch has stopped, the hour at

which it has stopped should be noted. In drowning, the watch commonly stops shortly after immersion. Grass, or plant-bearing soil underlying the body soon dries, turns yellow or brown and dies.

Radioactive Carbon: C_{14} accumulates in living organic matter. The C_{14} content of the organism is steadily maintained as long as it lives. **After death, the radioactivity gradually weakens taking about 5,600 years to reach half its initial activity.** A simple carbon compound, such as CO_2 or acetylene, or even carbon itself is prepared from the bones, and the radioactivity is estimated. In forensic work, radiocarbon dating is not useful as the technique cannot date bones less than a century old.

Tower of silence is used by Parsees to dispose of dead bodies.

PRESUMPTION OF DEATH

This condition has only legal importance. This question arises in cases of inheritance of property, or in obtaining insurance money, when a person is alleged to have been dead and the body is not found. Under the Indian Evidence Act, a person is presumed to be alive, if there is nothing to suggest the probability of death within thirty years. But if proof is produced that the same person has not been heard of for seven years by his friends and relatives, death is presumed.

PRESUMPTION OF SURVIVORSHIP

The question of presumption of survivorship may arise in connection with inheritance of property when two or more persons die in a common disaster, e.g., earthquake, shipwreck, plane crash, accident, etc.

COMMORIENTES: Persons who die together on the same occasion, where it cannot be ascertained who died first. The question may arise as to who survived longest and if no direct evidence on this is available, the question becomes one of presumption. The case is decided by the facts and evidence available. In the absence of such evidence, age, sex, constitution, nature and severity of injuries and the mode of death should be taken into consideration.

CHAPTER

8 Mechanical Injuries

Injury: An injury is any harm, illegally caused to any person in body, mind, reputation or property (S. 44, I.P.C.). A wound or injury is a loss of the natural continuity of any of the tissues of the living body. **Mechanical injuries (wounds) are injuries produced by physical violence.**

GENERAL PRINCIPLES: A wound is caused by a mechanical force which may be either a moving weapon or object, or the movement of the body itself. In the first case, the counterforce is provided by the inertia of the body, and in the second case by the rigidity of some stationary object against which he falls. A combination of these two events is seen in most cases. Due to the impact between the forward moving force and the counterforce, energy is transferred to the tissues of the body, which causes a change in their state of rest or motion. The human body contains many complex tissues which greatly vary in their physical properties, such as state of solidity, fluidity, density and elasticity, and because of this a change in the state of rest or motion of the body produced by a forceful impact does not affect the tissues uniformly. Some of the energy is spent in moving the body as a whole, but most of the energy may cause non-uniform motion of localised parts of the body, due to which the affected tissues will be subjected to compression or to traction strains or to a combination of both. All the body tissues, except those which contain gas, are resistant to compression, i.e., they resist force tending to reduce their volumes. Mechanical force does not cause compression of the tissue but causes their displacement and deformation, and traction strains are produced in the affected tissues. Such strains may be due to forces causing simple elongation of tissues, but they may be due to more complex mechanism, such as bending, torsion or shearing. The rigid tissues like bones resist deformation, but if the limit of their elasticity is exceeded, fracture occurs. The soft tissues are plastic, and as such, mechanical force alters their shape, which is limited by the cohesion between the tissue cells, connective and vascular tissue frameworks and capsules of organs. Soft tissues rupture when they are stretched beyond the limits of their tensile strength.

Classification of Injuries: Medical: (I) Mechanical or physical injuries: (A) Due to blunt force: (1) Abrasions. (2) Contusions. (3) Lacerations. (4) Fractures and dislocations. (B) Due to sharp-edged weapons: (1) Incised wounds. (2) Chop wounds. (3) Stab wounds. (C) Firearms: Firearm wounds.

(II) Thermal Injuries: (1) Due to cold: (A) Frostbite. (B) Trench foot. (C) Immersion foot. (2) Due to heat: (A) Burns. (B) Scalds.

(III) Chemical Injuries: (1) Corrosive acids. (2) Corrosive alkalis.

(IV) Explosions.

(V) Miscellaneous: Electricity, lightning, X-rays, radioactive substances, etc.

Legal: (1) Simple. (2) Grievous. (3) Dangerous.

Medicolegal: (1) Suicide. (2) Homicide. (3) Accident. (4) Fabricated. (5) Defence. (6) Self-inflicted. (7) Antemortem or post mortem.

ABRASIONS

An abrasion (gravel rash) (Plate 4-21) is a destruction of the skin or mucous membrane which usually involves the superficial layers of the epidermis only.

Mechanism: They are caused by friction against a rough surface or by compression, such as a lateral rubbing action by a blow, a fall on a rough surface, by being dragged in a vehicular accident, fingernails, thorns or teethbite. **Some pressure and movement by agent on the surface of the skin is essential. If sufficient friction is applied, partial or complete removal of the epidermis may occur, and the superficial layer of dermis is damaged.** Sometimes, full thickness of the skin may be damaged in places, but usually in an interrupted, irregular manner, and intact epidermis remains within the area of abrasion. **The rougher the surface and the more rapid the movement of the skin over it, the deeper is the injury.** Most abrasions are caused by a lateral

rubbing action. The exposed raw surface is covered by exudation of lymph and blood, which produces a protective covering known as a **scab or crust**. After death they become stiff, leathery and of a parchment like brown colour due to drying.

Features: Abrasions vary in size, depending on the extent of the body surface exposed to the abrading force. They are simple injuries, bleed slightly, heal rapidly without scar formation. Large abrasions can cause severe pain and bleeding. The size, situation, pattern and number of abrasions should be noted.

Types: Abrasions are of four types.

(1) Scratches: A scratch (linear abrasion) is an abrasion with length but no significant width, or a superficial incision depending on the agent (Plate 4-19 and 4-20). A scratch produced from the tip of the knife or razor can be called a **point scratch**. They are caused by a sharp object, not sharp enough to incise, but pointed enough to scratch, i.e., remove a portion of the skin's surface passing across the skin, such as fingernails, pin or thorn. A thorn or pin produces a narrow scratch which tails off. The surface layers of the skin are collected in front of the object, which leaves a clean area at the start and tags at the end **(Fig. 8-1)**.

Fingernail abrasions: When the skin is gripped in a static fashion, fingernail abrasions may be straight or curved, one to 2 mm broad, often about half to one cm. long, wide at the start, narrow at the end. They may be parallel linear scratches if the fingers are dragged down the skin. As the skin is put under lateral tension when it is indented by nails, it may distort, so that when the tension is released, the elasticity of the skin causes it to return to its original position, carrying the nail mark with it. The curve may then reverse to form either a straight line or a convexity, but this is always not true. Pointed nails are more likely than those with straight edges to give these paradoxical results. Longer, sharper fingernails cause linear, parallel, vertical scratches that may be several millimetres wide and placed a centimetre or two apart, if the fingers are dragged down the skin.

(2) Grazes (sliding, scraping or grinding abrasions): They are the most common type. **They occur when there is horizontal or near horizontal movement between the skin and some rough surface in contact with it. They show longitudinal parallel lines (grooves or furrows) with the epithelium collected at the ends of these lines, (which does not occur to a significant degree), and indicate the direction in which the force was applied.** The furrow may be broad at one end and narrow in the opposite direction **(Fig. 8-2)**. **Usually, the skin is uniformly detached at the start or may be serrated. The epidermis is scraped away, destroyed or detached.** These abrasions are commonly seen in a road accident. A glancing kick with a boot also produces a graze. Many abrasions extend into the dermis, because of the corrugations of the dermal papillae, and bleeding occurs. **An abrasion caused by violent rubbing against a broad rough surface, as in dragging over the ground is called brush burn or gravel rash**. It is a scraping injury over a large area. If a victim is struck a glancing blow with a rough object, such as a stone or a stick, brush burn will be caused **(Plate 4-22 to 4-24)**. **"Friction burn" (scuff or brush abrasion) is an extensive, superficial, reddened, excoriated area without serous ooze or bleeding and with little or no linear**

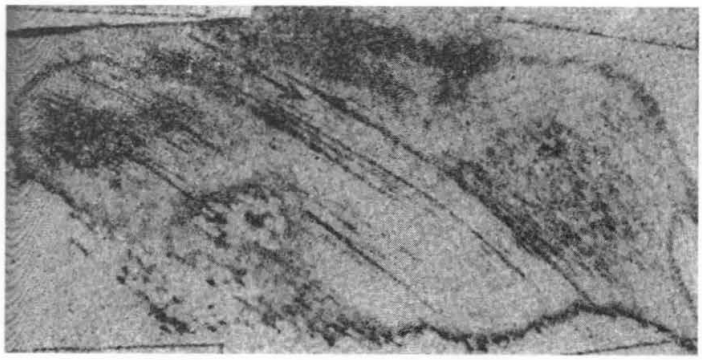


Fig. (8-1). Abrasion indicating direction of force.

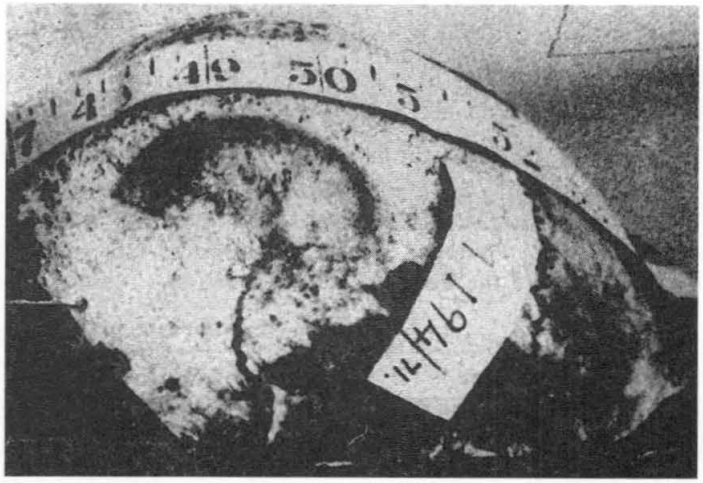


Fig. (8-2). Abrasion of the skull due to tearing force produced by the automobile wheel causing avulsion of the scalp and abrasion of skull bone.

local oedema. **If the impact is forcible, the dermis is damaged with an underlying bruise. If a person strikes a flat and relatively smooth surface, an abrasion can be produced which shows little or no linear markings,** as in traffic accidents.

particles of stone or sand are usually present, which may connect the injuries to the scene of crime. (6) Character and manner of injury may be known from its distribution. (a) In throttling, crescentic abrasions due to fingernails are found on the neck. (b) In smothering, abrasions may be seen around the mouth and nose. (c) In sexual assault, abrasions may be found on the breasts, genitals, inside of the thighs and around the anus. (d) Abrasions on the face or body of the assailant indicate a struggle. (e) Abrasions on the victim may show whether the fingernails of the assailant were long, irregular or even broken.

Differential Diagnosis: (1) Erosions of the skin produced by ants: Ants produce brown linear erosions with irregular margins of the superficial layers of the skin. They are most commonly found at mucocutaneous junctions, about the eyelids, nostrils, mouth and genitalia. They are also seen in the moist folds of the skin. Vital reaction is not seen. **(2) Excoriations of the skin by excreta:** In infants, slight inflammation with excoriation may be seen in the napkin area (groin and buttocks) at the time of the death (Nappy abrasions). After death, these areas become dry, depressed and parchment-like and the colour varies from pale-yellow to deep-copper. **(3) Pressure sores. (4) Drying of the skin of the scrotum** produces reddish-brown or yellow colouration resembling abrasion.

CONTUSIONS (BRUISES)

Mechanisms: A contusion is an effusion of blood into the tissues, due to the rupture of blood vessels, (venules and arterioles) due to blunt violence. **They are caused by blunt object,** such as fist, stone, stick, bar, whip, hammer, axe, wooden handle, poker, shod foot, boot, etc. **(Plate 5-26).**

Types: (1) Intradermal. (2) Subcutaneous. (3) Deep.

Features: The bruise is usually situated in the dermis and subcutaneous tissues, above the deep fascia, often in the fat layer. In contusion, there is a painful swelling and crushing or tearing of the subcutaneous tissues, usually without destruction of the skin. Bruises may be seen in association with abrasions (abraded contusions) or lacerations. A contusion is usually a superficial injury, but also occurs in deeper structures and viscera. A fresh bruise is usually tender and slightly raised above the surface of the skin, and even a deep-seated bruise shows some swelling when compared with the opposite limb or part of the body. **A bruise has lighter colour in the centre** because extravasated blood is pushed outward by the impact.

Size: Bruises vary in size from pinhead to large collections of blood in the tissues. When a large blood vessel is injured, a tumour-like mass called **haematoma** is formed. **Petechial bruises are finely mottled or stippled. If the petechiae become larger and confluent, they are called ecchymoses.** The size of a bruise is slightly larger than the surface of the agent which caused it, as blood continues to escape into the area. As a general rule, the greater the force of violence used, the more extensive will be the bruise.

Factors modifying size and shape of contusions:

(1) Condition and Type of Tissue: If the part is vascular and loose, such as face, vulva, scrotum, a slight degree of violence may cause a large bruise. Bruising is more marked on tissues overlying bone which acts as anvil, with the skin between the bone and the inflicting force. Chronic alcoholics bruise easily. **If the tissues are strongly supported, and contain firm fibrous tissues and covered by thick dermis, such as abdomen, back, scalp, palms and soles, a blow of moderate violence may produce a comparatively small bruise.** In boxers and athletes, bruising is much less because of good muscle tone (Resilient areas, such as the abdominal wall and buttock, bruiseless). **Bruising is not seen** (a) if the injured part is thickly covered, (b) part of the body is yielding, (c) if the weapon used is a yielding one, such as a sandbag. (d) if the pressure is continued until death occurred.

(2) Age: Children bruise more easily, because of softer tissues and delicate skin, and old persons bruise easily because of loss of flesh and cardiovascular changes.

(3) Sex: Women bruise more easily than men, because tissues are more delicate and subcutaneous fat is more. Fat people bruise easily. A slight pressure with the fingers on the arm of a woman, especially if she is obese and not accustomed to work or exercise, may produce a definite bruise.

(4) Colour of Skin: Bruising is more clearly seen in fair-skinned persons than those with dark skin in whom they may be better felt than seen. **The areas of extravasated blood appear darker even**

on heavily pigmented Negroid skin. Contusions appear much more clear in black and white or colour photographs.

(5) Natural Disease: When the vessels are diseased as in arteriosclerosis, bruising occurs very easily and may even result from coughing or slight exertion. In children, small bruises may be caused by the violent coughing as in whooping cough. In purpura haemorrhagica, leukaemia, haemophilia, scurvy, vitamin K and prothrombin deficiency, and in phosphorus poisoning, prominent bruising occurs following minor trauma.

(6) Gravity Shifting of the Blood: Bruises do not always appear at the site of impact. Blood escaping from deep torn vessels may be prevented from reaching the skin at the point of impact because of fascial plane arrangements at the injured area. **The bruise may appear at a relatively distant place, where the fascial pattern permits it to reach the surface (ectopic bruising or percolated contusion).**

Example: (1) Haemorrhages in the soft tissues around the eyes and in the eyelids (spectacle haematoma; black eye), may be caused by (a) direct trauma, such as a punch in the eye **(Plate 5-27)**, (b) blunt impact to the forehead, the blood gravitating downwards over the supraorbital bridge, (c) fracture of the floor of the anterior fossa of the skull. (2) In fracture of the jaw, a bruise may appear in the neck. (3) A bruise behind the ear may indicate a basal fracture, rather than a direct blow behind the ear. **(Battle's sign)**. (4) In fracture of the pelvis, a bruise may appear in the thigh. (5) In fracture of the femur, a bruise may appear on the outer side of the lower part of the thigh. (6) A kick on the calf of the leg may appear as a bruise around the ankle.

Patterned Bruising: A patterned contusion is one in which the size and shape, mirror a portion of the object which caused it. A bruise is usually round, but it may indicate the nature of the weapon, especially when death occurs soon after infliction of injury. If the person is living, this pattern may become obscure as the area of bruising tends to extend and merge with adjacent structures. (1) A blow from a solid body, such as a hammer or the closed fist usually produces a rounded bruise. (2) Bruises made by the end of a thick stick may be round, but if any length of the stick hits the body, they are elongated and irregular. (3) A blow with a rod, a stick or a whip produces two parallel, linear haemorrhages (railway line or tramline type). The intervening skin is unchanged, because the rod forcibly dents the tissues inwards and momentarily stretches each side of the dent. This causes rupture of vessels with a line of bruising, whereas the base of the dent becomes compressed and the vessels are not injured. When the rod is removed and the skin comes back to its normal position, the two sides of the depression remain as contused lines **(Plate 5-28 to 5-30)**. In a bruise produced by a long rigid weapon, e.g., stick, the edges of the bruise may be irregular and the width may be greater due to infiltration of blood in the surrounding tissues along the edges of the bruise. (4) When the body is struck by a broad flat weapon, such as a plank, the edges of the plank may cause parallel bruises in the skin, separated by normal tissue. (5) Bruises caused by blows from whips are elongated, curve over prominences, and may partially encircle a limb or the body. They are seen as two parallel lines, the distance between which is roughly equal to the diameter of the whip. (6) Bruises made by pliable canes are similar to those due to whip, but never encircle a limb, or curve round the sides of the body. (7) Bruises from straps, belts or chains leave a definite imprint. (8) A woven, spiral or plaited ligature may sometimes produce a patterned bruise. (9) Contact injuries from firearms may produce abrasion with bruising, indicating the outline of the muzzle of the weapon. (10) Patterned bruising is seen in motor car accidents. (11) Suction or biting on the sides of the neck or the breasts, during erotic love-making or sexual intercourse produces elliptical patterned bruise. (12) If a violent blow is struck on a clothed area of the body, or if the clothing is grabbed and twisted over the skin, petechial haemorrhages occur within the skin reproducing the texture of clothing.

Delayed Bruising: A superficial bruise appears immediately as a dark-red swelling. A deep bruise may take several hours, or one or two days to appear, and deeper extravasation of blood may never appear. Therefore, one more examination should be carried out 48 hours after the first examination, to note bruises of slower development **(come-out bruise). Occasionally, when an injury is produced before death, the bruise may appear some time after death, due to further escape of blood from the ruptured vessels due to gravitation,** mainly due to percolation

and rapid haemolysis of stagnant blood, the pigment diffusing locally and producing a stain on the surface. This may explain the difference of opinion between two observers, who have examined the person or the body at different times.

Deep Tissue and Organ Contusions: All organs can be contused. Deep contusions are clearly seen during autopsy, as the blood is drained from blood vessels and also due to postmortem autolytic changes. (1) Contusions in vital centres, e.g., which control respiration and blood pressure can be fatal, even when very small. (2) A small contusion of the heart can cause serious disturbance of normal rhythm or stoppage of cardiac action and death. Large contusions often prevent adequate cardiac emptying and lead to heart failure. (3) Contusions of other organs may cause rupture of that organ with slow or rapid bleeding into the body cavity and may cause death.

The Age of Bruise: A bruise heals by destruction and removal of the extravasated blood. The haemoglobin is broken down into haemosiderin, haematoidin and bilirubin by the action of enzymes. **The colour change starts at the periphery and extends to the centre. The rate of colour change is quite variable, not only between persons, but in the same person and from bruise to bruise.** Factors affecting colour of contusions include: (1) Depth of bleeding. (2) Amount of bleeding. (3) Environmental lighting, and (4) Overlying skin colour. Bruises in children change colour rapidly and may be completely absorbed in a few days. **The doctor can only state that the appearance (colour) of a contusion is consistent with its having been caused so many days back. The colour changes seen:**

At first: Red.

Few hours to 3 days: Blue.

4th day: Bluish-black to brown (haemosiderin).

5 to 6 days: Greenish (haematoidin).

7 to 12 days: Yellow (bilirubin).

2 weeks: Normal.

Antemortem and Postmortem Bruising: In antemortem bruising, there is swelling, damage to epithelium, extravasation, coagulation, and infiltration of the tissue with blood and colour changes. These signs are absent in postmortem bruises. **The margins of postmortem bruises are usually quite sharply defined and antemortem bruises are less sharp or indistinct for the greater part, indicating vital reaction in the damaged tissue. Appreciable bruising does not occur two minutes after death due to arrest of circulation.**

Autopsy: At autopsy, bruises may not be readily detected or they may be obscured by patches of postmortem lividity, or by dark colour of the skin. They may be detected by parallel incisions through the skin. **Deep bruises are detected by deep incisions made into the muscles (Plate 6-31 and 6-32).** The extent of bruising and the injury to the underlying soft tissues and muscles can be seen by dissecting the skin away from the underlying fat and muscle fascia. When in doubt, a portion must be taken for microscopy **(Table 8-2)**.

Medicolegal Importance: (1) They give an idea about the site of impact. (2) The degree of violence may be determined from their size. (3) Patterned bruises may connect the victim and the object or weapon, e.g., whips, chains, canes, ligature, vehicle, etc. (4) The age of the injury can be determined. (5) In open wounds, dirt, dust, grease or particles of stone or sand are usually present, which may connect the injuries to the scene of crime. (6) Character and manner of injury may be known from its distribution. (a) When the arms are grasped, there may be 3 or 4 bruises on one side and one larger bruise on the opposite side, from the fingers and thumb respectively, indicating the position of the assailant in front of or behind the victim. (b) Bruising of the arms in a victim may indicate restraint. (c) Bruising of the shoulder-blades indicates firm pressure on the body against the ground or other resisting surface. (d) In manual strangulation, the position and number of bruises and nail marks may give an indication of the method of attack or the position of the assailant. (e) Bruising of thighs especially inner aspect, and of genitalia indicates rape.

Bruises are of less value than abrasions because: (1) Their size may not correspond to the size of the weapon. (2) They may become visible several hours or even one to two days after the injury. (3) They may appear away from the actual site of injury. (4) They do not indicate the direction in which the force was applied.

Complications: (1) **A contusion may contain 20 to 30 ml. of blood or even more. Multiple**

Table (8-2). Difference between P.M. hypostasis and bruise

Trait	P.M. Hypostasis	Bruise
(1) Cause:	Due to vessels in the dermis distended with blood.	Due to ruptured vessels which may be superficial or deep.
(2) Site:	Occurs over extensive area of the most dependent parts.	Occurs at the site of and surrounding the injury, anywhere on the body.
(3) Situation:	Lies in the dermis.	Lies under the epidermis in the interstices or still deeper.
(4) Appearance:	No elevation of the involved area.	Often swollen because of extravasated blood and oedema.
(5) Epidermis:	Not abraded.	May be abraded.
(6) Margins:	Clearly defined.	Merge with the surrounding area.
(7) Colour:	Uniform bluish-purple.	Old bruises are of different colour. Fresh bruises may appear more intense than the adjacent hypostatic area.
(8) Incision:	On incision, blood is seen in blood vessels, which can be easily washed away. Subcutaneous tissues are pale.	Shows extravasation of blood into the surrounding tissues which is firmly clotted and cannot be washed by gentle stream of water. Subcutaneous tissues are deep reddish-black.
(9) Effect of pressure:	Absent in areas of the body which are under slight pressure.	Little lighter over the area of pressure or support.

contusions can cause death from shock and internal haemorrhage. (2) Gangrene and death of tissue can result. (3) The pooled blood can serve as a good site for bacterial growth, especially by clostridial group. (4) Rarely in severe sudden compression of the subcutaneous tissue, pulmonary fat embolism may occur.

Artificial Bruises: Some irritant substances, when applied to skin produce injuries, which simulate bruises (**Table 8-3 and Plate 6-33**). They are produced to make a false charge of assault.

CIRCUMSTANCES OF INJURIES: **Accidental bruises are very common and may be seen on prominences, such as the forehead, nose, elbows and knees. Self-inflicted bruises are rare as they are painful. They are seen usually on the head, especially in a hysterical individual or the insane. Homicidal bruises may be seen on any part of the body. Kicking and jumping on a person combined is known as "STOMPING". It is not possible to differentiate an injury caused by a fist or weapon and a fall. Contamination of the wound should be looked for.**

LACERATIONS

Lacerations are tears or splits of skin, mucous membrane, muscle or internal organs produced by application of blunt object to broad area of the body, which crushed or stretched tissues beyond the limits of their elasticity. They are also called tears or ruptures.

Causes: They are caused by blows from blunt objects, by falls on hard surfaces, by machinery, traffic accidents, etc. If the force produces bleeding into adjacent tissues, the injury is a **'contused-laceration' or 'bruised-tear'.** If the margins are abraded, it is called **'abraded laceration'** or **'scraped tear'**. If the blunt force produces extensive bruising and laceration of deeper tissues it is called **crushing injury.**

Mechanism: The object causing a lacerated wound crushes and stretches a broad area of skin, which then splits in the centre. The edges are irregular and rough, because of the crushing and tearing nature of the blunt trauma. Frequently, the skin at the margins are abraded due to the flatter portion of the striking object rubbing against the skin, as it is indented by the forceful blow. The margins are contused due to the bleeding into the tissues caused by trauma. If a blunt object, e.g. a bottle strikes the skin surface, the edge at the point of impact will be sharp and turned inwards, whereas the other edge may be everted exposing the hair follicles in the depths. In an impact over the scalp, external laceration may

Table (8-3). Difference between true bruise and artificial bruise

Trait	Artificial bruise	True bruise
(1) Cause:	Juice of marking nut, calotropis or Plumbago rosea.	Trauma.
(2) Site:	Exposed accessible parts.	Anywhere.
(3) Colour:	Dark-brown.	Typical colour changes.
(4) Shape:	Irregular.	Usually round.
(5) Margins:	Well-defined and regular, covered with small vesicles.	Not well-defined, diffuse and irregular; no vesicles.
(6) Redness and inflammation:	Seen in the surrounding skin.	Seen in the site.
(7) Contents:	Acrid serum.	Extravasated blood.
(8) Itching:	Present.	Absent.
(9) Vesicles:	May be found on fingertips and on other parts of the body due to scratching.	Absent.
(10) Chemical tests:	Positive for the chemical.	Negative.

not occur due to the hair, but inner layers of scalp may be lacerated. If the instrument is padded or has a broad striking surface, severe fractures of the skull may occur without external laceration.

Lacerations of the internal organs may be caused by: (1) direct injury of the viscera by fragments of fractured bone (**Plate 7-38**), (2) development of traction shears or strain shears in the viscera, (3) stretching of the visceral attachments, and (4) hydrostatic forces.

Types: (1) Split Lacerations: Splitting occurs by crushing of the skin between two hard objects. Scalp lacerations occur due to the tissues being crushed between skull and some hard object, such as the ground or a blunt instrument (**Fig. 8-4 , Plate 6-34 and 6-35**).

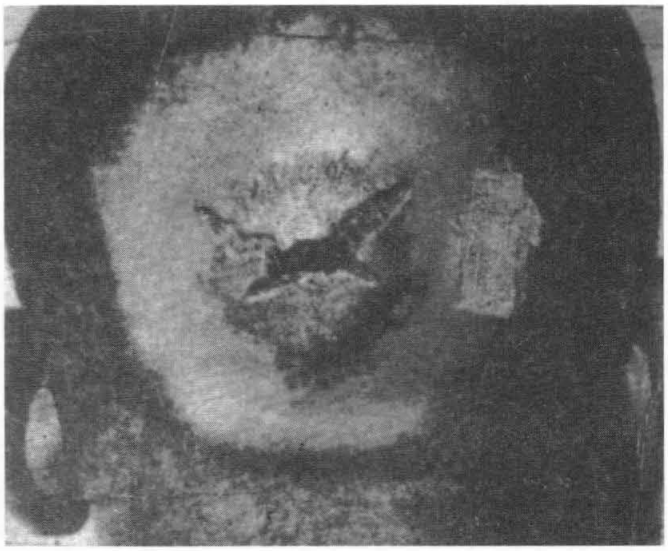


Fig. (8-4). Laceration of scalp (star-shaped).

Incised-like or Incised-looking Wounds: Lacerations produced without excessive skin crushing, may have relatively sharp margins. Blunt force on areas where the skin is close to bone, and the subcutaneous tissues are scanty, may produce a wound which by linear splitting of the tissues, may look like incised wound. The sites are the scalp, eyebrows, cheek bones, lower jaw, iliac crest, perineum, and shin. A wound produced by a fall on the knee or elbow with the limb flexed, and by a broken glass, or sharp stone also looks like incised wound (**Table 8-5**).

(2) Stretch Lacerations: Overstretching of the skin, if it is fixed, will cause laceration. There is localised pressure with pull which increases until tearing occurs and produces the "flap". This is seen in the running over by a motor vehicle, and the flap may indicate the direction of the vehicle. They can occur from kicking, and also when sudden deformity of a bone occurs after fracture, making it compound.

(3) Avulsion: An avulsion is a laceration produced by sufficient force (shearing force) delivered at an acute angle to detach (tear off) a portion of a traumatised surface or viscus from its attachments. The shearing and grinding force by a weight, such as lorry wheel passing over a limb, may produce separation of the skin from the underlying tissues (avulsion) over a relatively large area. This is called "flaying" (Fig. 8-5 and

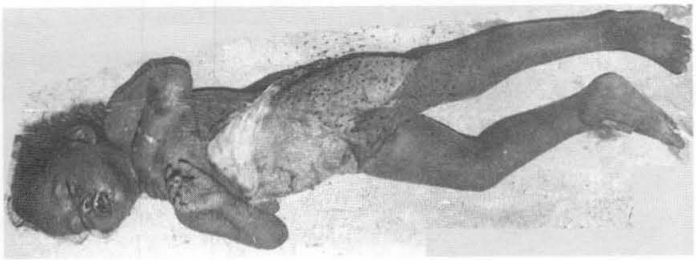


Fig. (8-5). Avulsion caused by running over of the wheel of automobile. The skin is torn away in a flap due to the grinding action of the wheel.

Plate 6-36). The underlying muscles are crushed, and the bones may be fractured. The separated skin may show extensive abrasions from the rotating frictional effect of the tyre, but one portion is still in continuity with adjacent skin. Internal organs can be avulsed or torn off in part or completely from their attachments. Avulsion of scalp is also caused by traction from hair being trapped in machinery.

(4) Tears: Tearing of the skin and tissues can occur from impact by or against irregular objects, such as door handle of car. It may be caused by blows by broken glass, or a fall over a rough projected object. A tear is deeper at the starting point than at the termination. This is another form of overstretching.

(5) Cut Lacerations: Cut lacerations may be **produced by not so sharp edges of a heavy cutting weapon,** such as axe, hatchet, chopper, etc. The margins are usually abraded with bruising.

Characters: (1) Margins are irregular, ragged and uneven and their ends are pointed or blunt, but show minute tears in the margins. The edges of laceration, especially over a bony area, e.g. skull are undermined due to the crushing and tearing force of the impact. **Tearing at the ends of lacerations, at angles diverging from the main laceration itself, so-called swallow tails,** are frequently noted. (2) Bruising is seen either in the skin or the subcutaneous tissues around the wound. (3) Depth varies according to the thickness of the soft parts at the site of the injury and degree of force applied. (4) Deeper tissues are unevenly divided with tags of tissue at the bottom of the wound bridging across the margin. **Tissue bridges** consist of nerves, blood vessels and elastic and connective tissue fibres. (5) Hair bulbs are crushed. (6) Hair and epidermal tags may be driven deeply into the wound. (7) Haemorrhage is less because the arteries are crushed and torn across irregularly, and thus retract and the blood clots readily. (8) Foreign matter may be found in the wound. (9) The shape and size may not correspond with the weapon or object which produced it. A laceration is usually curved; the convexity of the curve points towards the direction of application of force. (a) A blunt round end may cause stellate laceration. (b) A blunt object with an edge, such as hammer head, may cause crescentic laceration. (c) Long, thin objects, such as pipes, tend to produce linear lacerations, while objects with flat surfaces produce irregular, ragged, or Y-shaped lacerations. (10) Direction: The skin on side of wound opposite to direction of motion is usually torn free or undermined for a variable distance. The other side, i.e. the side from which the blow was delivered, will be abraded and bevelled.

Age: Age determination of laceration is difficult unless there are clear signs of healing, such as granulation tissue, fibroblast ingrowth, or organising infiltrate.

Antemortem lacerations show bruising of margins, vital reaction, eversion, blood-staining of margins, and gaping of the margins.

Complications: (1) Laceration of an internal organ may cause severe or even fatal bleeding. Multiple lacerations, involving only the skin and subcutaneous tissue, each causing some haemorrhage, may combine to cause shock and death. (2) Pulmonary or systemic fat embolism may occur due to crushing of subcutaneous tissue. (3) Infection.

Medicolegal Importance: (1) The type of laceration may indicate the cause of the injury and the shape of the blunt weapon. (2) Foreign bodies found in the wound may indicate the circumstances in which the crime has been committed. (3) The age of the injury can be determined.

CIRCUMSTANCES OF INJURIES: Suicidal lacerations are very rare, as they are painful. Accidental lacerations are usually situated on the exposed parts of the body and mostly on the same side. In the case of fall on the head, the abraded scalp surface will be circular and completely surrounds the laceration. Homicidal wounds are usually seen on the head (Fig. 8-4).

Combination of Abrasions, Contusions and Lacerations: Abrasions, contusions and lacerations are frequently seen together, or as integral parts of one another. The same object may cause a contusion with one blow, a laceration with second, and an abrasion

with a third. Sometimes, all three types of injury may result from a single blow. Sometimes, an imprint may result from an object, and it may be difficult to determine whether the imprint is primarily an abrasion or a contusion. Abrasions, contusions and lacerations are the external visible evidence of blunt impact injury which may be the only external sign of deeper and sometimes fatal injury of deeper lying structures.

INCISED WOUNDS

An incised wound (cut, slice) is a clean cut through the tissues, which is longer than it is deep. It has length rather than depth and tends to gape. It is produced by the pressure and friction against the tissue by an object having a sharp-cutting edge, such as knife, razor, scalpel, scissors, sickle, glass, cleaver, sword, etc. In this, the force is applied over a very narrow area, corresponding with the cutting edge of the blade **(Plate 7-39 and 7-40). Some injuries cannot be divided clearly into stab or incised wounds, but may exhibit features of both.**

Causes: (1) Striking the body with the edge of sharpcutting weapon, against the body surface, (2) by drawing the weapon, and (3) by using the weapon, like a saw.

Characters: (1) **Margins: The margins are cleancut, well-defined and usually everted.** The edges may be inverted if a thin layer of muscle fibres is adherent to the skin as in the scrotum. The edges are free from contusions and abrasions. The depth of the wound will not show any tissue bridging. (2) **Length: The length is greater than its width and depth, and has no relation to the cutting edge of the weapon,** for it may be drawn to any distance. When the skin becomes folded under the cutting edge of the weapon, a single movement of the weapon may produce a series of incised wounds, separated one from another by bridges of normal skin. (3) **Width: The width is greater than the thickness of the edge of the weapon causing** it, due to retraction of the divided tissues. Twisting of the blade tends to make the wound wider than the weapon. (4) **Shape: It is usually spindle-shaped** due to greater retraction of the edges in the centre. Gaping is greater if the underlying muscle fibres have been cut transversely or obliquely and less when cut longitudinally. Incised wounds crossing irregular surfaces may be irregular in depth, but will be linear. The wound may take zig-zag course, if the skin is loosely attached as in axillary fold, because the skin is pushed in front of the blade before it is cut. If the blade is curved, the edges will be crescentic. If the surface is convex, such as occipital region or buttocks, the straight-bladed weapon may also produce a crescentic wound. (5) **Haemorrhage:** As the vessels are cut cleanly, the haemorrhage is more. Spurting of blood occurs if an artery is cut. (6) **Direction:** Incised wounds are deeper at their beginning because more pressure is exerted on the knife at this point. This is known as the **head of the wound**. Towards the end of the cut, the wound becomes increasingly shallow, till finally as the knife leaves the tissues, the skin alone is cut. This is known as the **tailing of the wound**, and indicates the direction in which the cut was made. (7) **Bevelling cut:** If the blade of weapon enters obliquely, the wound will have a bevelled, margin on one side with under mining (undercutting), so that subcutaneous tissue is visible, indicating the direction from which the blade entered, and if the blade is nearly horizontal, a flap wound is caused. **Bevelling (overhanging or undermining) can be produced by sharp weapon only. It is usually homicidal** and may indicate the relative position of the assailant and the victim.

Age of Incised Wound: In an uncomplicated wound healing occurs as follows: **Fresh:** Haematoma formation. **12 hours**: The edges are red, swollen and adherent with blood and lymph; leucocytic infiltration. **24 hours:** A continuous layer of endothelial cells covers the surface; overlying this a crust or scab of dried clot is seen.

Histopathology: Few minutes: Dilatation of capillaries and margination and emigration of neutrophils, reactive changes in the tissue histiocytes and swelling of the vascular endothelium. 12 hours: Reactive changes occur in the fibroblasts at the site of injury within few hours; monocytes appear in the exudate. 15 hours: Monocytes begin to undergo mitotic division. 24 hours: Epithelium begins to grow at the edges. Vascular buds begin to form. 72 hours: Vascularised granulation tissue is formed. 4 to 5 days: New fibrils are formed. One Week: Scar formation in small wounds.

Wounds by Glass: Wounds produced by glass are lacerated, but can resemble incised and stab wounds **(Plate 7-37)**. If a small sharp-pointed piece

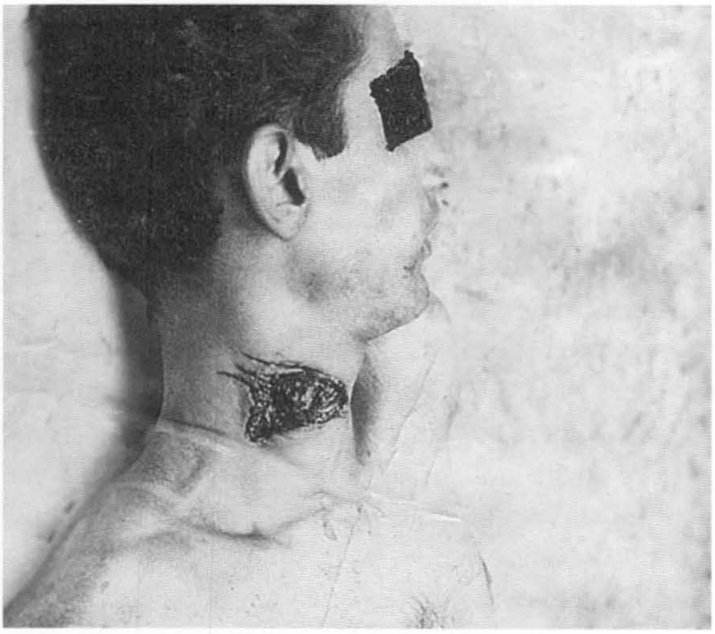


Fig. (8-6). Suicidal cut-throat with hesitation marks.

of glass enters by its point, the wound has a stab-like appearance. **Margins of the wound will almost always show tiny side-cuts due to irregularities of the glass**. Particles of glass may be found in the wound. Assault with broken glasses or bottles show multiple, irregular incised-type wounds of variable depth and severity.

Medicolegal Importance: (1) They indicate the nature of weapon (sharp-edged). (2) They give an idea about the site of impact and direction of the force. (3) The age of the injury can be determined. (4) Position and character of wounds may indicate mode of production, i.e., suicide, accident or homicide.

Features of suicidal wounds (Table 8-4): (1) They are multiple and parallel or nearly so, in any one area. (2) They are uniform in depth and direction. (3) They are relatively trivial. (4) The fatal wounds are present on several limited, easily reached areas of the body, such as front of the neck, wrists, groin, and occasionally on the back of legs or on chest (**Fig. 8-6 and Plate 7-41 and 7-42**). (5) They avoid vital and sensitive areas like eyes, lips, nose and ears. (6) **Hesitation marks or tentative cuts or trial wounds:** They are cuts which are multiple, small and superficial often involving only the skin and are seen at the beginning of the incised wound. When a safety razor blade is used, **unintentional cuts** are found on the fingers where the blade has been gripped.

CIRCUMSTANCES OF INJURIES

Suicidal incised wounds of extremities are usually found on the flexor surface of the wrists, elbows and in some cases on the groins, thighs, ankles, knees, or on the radial border of the forearm. Suicidal wounds of chest are usually on the left side and directed downwards and inwards. A person who commits suicide, exposes his body by opening his clothes and then inflicts wounds. If the person dies, the wound should be dissected to determine injury to major blood vessels. Usually, the victim hyperextends the wrist before making the cut, which causes the radial artery to slip at the lower end of the radius, which escapes injury. Flexor tendons may be cut.

Homicidal wounds of the chest are usually present over a wider area and are more horizontal. They may be directed from below upwards, which is rarely seen in suicidal wounds. Incised wounds situated on the back, or in such a position as cannot be easily reached by a suicide are homicidal. Incised wounds on nose, ears and genitals are usually homicidal, and are inflicted on account of jealousy or revenge (in cases of adultery) causing disfiguration. Cut-throat wounds cause death from haemorrhage, air embolism or inhalation of effused blood into the respiratory tract (Table 8-4).

Accidental incised wounds may be caused by falling upon a sharp-cutting weapon held in the hand, or upon a sharp-pointed object, or by a sharp piece of broken glass. They may be situated anywhere on the body but are commonly seen about the hands (Table 8-6).

In a decomposed body, it is difficult to differentiate a laceration from an incised wound. When a body with incised, stab, or lacerated wounds is immersed in water soon after infliction of injuries, it cannot be made out whether they are antemortem or postmortem.

CHOP WOUNDS: They are deep gaping wounds caused by a blow with the sharp-cutting edge of a fairly heavy weapon, like a hatchet, an axe, sword, broad heavy knife, or a meat cleaver. **The dimensions of the wound correspond to cross-section of penetrating portion of blade.** The margins are sharp and may show slight abrasion and bruising with marked destruction of underlying organs. If the edge is blunt, the margins are ragged and bruised. Undermining occurs in the direction towards which the chop is made. When the whole blade strikes the body at the same time, the depth may be same throughout the wound. Usually the lower end (heel) of the axe strikes the surface first, which produces a

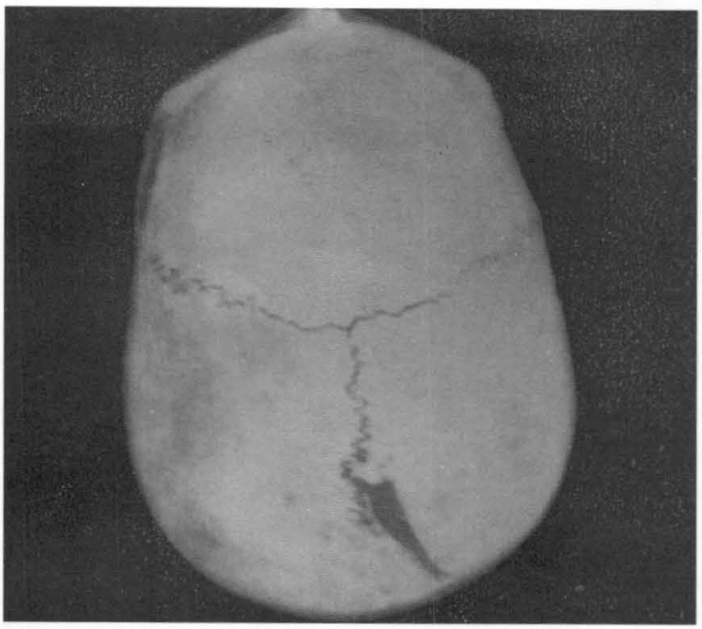


Fig. (8-7). Cut injury of the skull. The wide gap anteriorly is caused by the heel of the axe in a frontal attack.

deeper wound than the upper (toe) end wound. The deeper end indicates the position of the assailant. If the extremities are attacked, there may be complete or incomplete amputation of the fingers or other bones, and the joints may be separated or disarticulated. Wounds on the head and trunk are usually associated with injuries to important structures and are fatal (**Fig. 8-7 and Plate 8-43 to 8-45**). The cranium may be depressed, but if the blow is a glancing one, a piece of the skull may be removed. The neck may be almost completely separated.

Circumstances of injuries: Most of these injuries are homicidal and usually inflicted on the head, face, neck, shoulders and extremities. Accidental injuries are caused by power fans, band saws or ship or airplane propellers, which may lacerate the soft tissues extensively or amputate parts of the body. Suicidal injuries are rare.

STAB OR PUNCTURE WOUNDS

A stab wound is produced when force is delivered along the long axis of a narrow or pointed object, such as knife, dagger, nail, needle, spear, arrow, screwdriver, etc. **into the depths of the body, that is deeper than its length and width on surface of skin**. This can occur by driving the object into the body, or from the body's pressing or falling against the object (**Table 8-5**).

Types: (1) **Puncture wounds** when muscles or viscera are involved. (2) **Penetrating wounds**, when they enter a cavity of body. (3) When the weapon enters the body on one side, and comes out on the other side, **perforating wounds or through-and-through puncture wounds** are produced. The wound of entry is larger with inverted edges, and the wound of exit is smaller with everted edges, due to tapering of blade.

Characters: (1) Margins: The edges of the wound are clean-cut and everted. The margins can be inverted when the wound is situated over the fatty area, such as protuberant abdomen or gluteal region. There is usually no abrasion or bruising of the margins, but in full penetration of the blade, bruising (hiltmark) may be produced by the hilt or hilt-guard striking the skin. The margins may be abraded, and ragged if the cutting edge is blunt.

(2) Length: The length of the wound is slightly less than the width of the weapon up to which it has been driven in, because of stretching of the skin. For measuring the length of the stab wound, the edges of the wound should be brought together. If the instrument is thrust in, and is then completely withdrawn with the cutting edge dragging against one end, the wound would be extended superficially, producing a tail (**Fig. 8-8 and Plate 9-50**).

(3) Width: In most cases, the wound gapes across the centre, to form a long ellipse. **The maximum possible width of knife blade can be approximately determined if the edges of a gaping wound are brought together.** Elasticity or laxness of skin can change width by 1 to 2 mm. The opening may enlarge by backward, forward or a lateral movement of the weapon. The wound will gape, if a muscle is cut across.

(4) Depth: The depth (length of track) is greater than the width and length of the external injury. The depth of a stab wound is usually equal to, or less than the length of the blade, that is used in producing it, **but on yielding surfaces like the anterior abdominal wall, the depth of the wound may be greater, because the force of the thrust may press the tissues underneath.** In young persons, the chest is mobile and may be compressed during the stabbing (**Fig. 8-9**). **The breasts, buttocks and thighs are indented by a full thrust, and the depth**

Table (8-4). Difference between suicidal and homicidal cut-throat wounds

Trait	Suicidal wounds	Homicidal wounds
(1) Situation:	Left side of the neck and passing across the front of the throat; rarely on both sides.	Usually on the sides.
(2) Level:	High; above the thyroid cartilage.	Low; on or below the thyroid cartilage.
(3) Direction:	Above downwards and from left to right in right-handed person. Sometimes horizontal cuts are seen which do not show variation in depth at either end.	Transverse or from below upwards. If attacked from behind, it may resemble suicidal wound.
(4) Number of wounds:	Multiple, may be 20 or 30, superficial, parallel and merged with the main wound; rarely single.	Multiple, cross each other at a deep level; not repeated in depths of the main wound.
(5) Edges:	Usually ragged due to overlapping of multiple superficial incisions.	Sharp and clean-cut; bevelling may be seen.
(6) Hesitation cuts:	Present.	Absent.
(7) Tailing:	Present.	Absent.
(8) Severity:	Usually less severe. One wound may be extremely deep, extending to the cervical vertebrae, but sometimes two or three may be severe.	More severe; all the tissues including the vertebrae may be cut.
(9) Wounds in other parts of the body:	Often present across wrists, groin, thighs, ankles, or knees; and rarely on the back of the neck.	No wounds on wrists, etc., but severe injuries usually on the head and neck.
(10) Defence wounds:	Absent; unintentional cuts may be found on the fingers if a blade has been used.	Present.
(11) Hands:	Weapon may be firmly grasped due to cadaveric spasm.	Fragments of clothing, hair, etc., may be grasped.
(12) Weapon:	Usually present.	Usually absent.
(13) Vessels:	As head is thrown back, carotid artery is drawn beneath sternomastoid and against the spine and usually escapes injury.	Jugular vein and carotid artery are likely to be cut.
(14) Bloodstains:	If standing, stains on the mirror and on front of body and clothes, running from above downwards, and splashes on feet.	If asleep, blood runs down on both sides of the neck and collects behind the neck and shoulders; stains found on both palms, for the victim attempts to cover the wound.
(15) Clothes:	Not cut or damaged.	May be cut corresponding in position to those in the body; disarrangement, tears and loss of buttons.
(16) Circumstantial evidence:	Quiet place, such as bedroom or locked bathroom; usually stands in front of a mirror in order to direct the hand better; suicidal note or farewell letter may be found.	Disturbance at the scene, such as disarrangement of furniture in a room; trampling and crushing of vegetable matter and shrubs, or confused footprints outside.

Fig. (8-8). Multiple stab injuries some of them showing tailing.

Fig. (8-9). Assessment of depth of penetration of a stab wound.

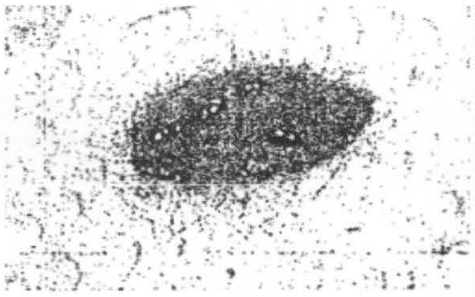


Fig. (8-10). Boat-shaped stab wound produced by a single-edged blade.

of the wound may exceed the length of the weapon. Many of the internal organs are not fixed but have a variable degree of mobility. This should be taken into account when estimating the depth of wound. **After the withdrawal of the weapon, the wound tends to close by expansion of the tissues along the track. The skin is most resistant to knife penetration. The sharpness of the extreme tip of the knife is the most important factor in skin penetration.** Once the tip has perforated the skin, the cutting edge is of little importance, and the rest of the blade will pass into the body easily, without any further force being applied, unless obstructed by bone or cartilage. **A thin, slender, double-edged knife will penetrate more deeply than an equally sharp, wide, single-edged blade inserted with the same force.** A blunt-pointed instrument requires considerable force to puncture the skin and penetrate the soft tissues. **Stretched skin is easier to penetrate than lax skin. When the instrument strikes the skin at right angle, it usually penetrates more deeply than when it strikes from some acute angle.**

(5) Shape: The size and shape of a stab wound in the skin is dependent on the type of implement, cutting surface, sharpness, width and shape of the weapon, the direction of thrust, the movement of the blade in the wound, cleavage direction, the movement of the person stabbed, and the condition of tension or relaxation of the skin **(Fig. 8-14)**.

Lines of cleavage of Langer: The pattern of fibre arrangement of the dense feltwork of intimately intermingled dermal collagen and elastic fibres is called the cleavage direction or **lines of cleavage** of the skin, and their linear representation on the skin are called **Langer's lines**, which are almost same in all persons. **Cleavage lines in the dermal layers of skin are mostly arranged in parallel rows.** In the extremities, they tend to run longitudinally, in the neck and trunk circumferentially **(Fig. 8-11)**. A stab wound which cuts through the cleavage lines transversely or obliquely will gape **(Plate 9-49)**.

Weapon causing stab: (Figs 8-12 to 8.14) The shape of the wound usually corresponds to the weapon used, but the shape of the wounds made by the same weapon may differ on different parts of the body. (a) If a single-edged weapon is used, the surface wound will be triangular or wedge-shaped, and one angle of the wound will be sharp, the other blunt or torn **(Fig. 8-10 and Plate 8-46 to 8-48)**. Blunt end of the wound may have small splits (often in two places) in the skin, so-called **"fishtailing"**, if the back edge of the blade is stout. When the blade enters parallel to cleavage lines, usually a single-edged weapon produces two sharply marginated acute angles, because the initial penetration by the knife-point, first produces a skin defect with sharp angles at each end. Many single-edged knives have a cutting edge on both sides at the tip. The initial thrust into the skin will produce a double-pointed wound, and as the blade passes through the skin, if it is pulled down slightly, the blunt back portion of the knife will not come in contact with the skin. In some single-edged knives, both ends are blunt. This is caused if the knife penetrates to the full length up to the guard,

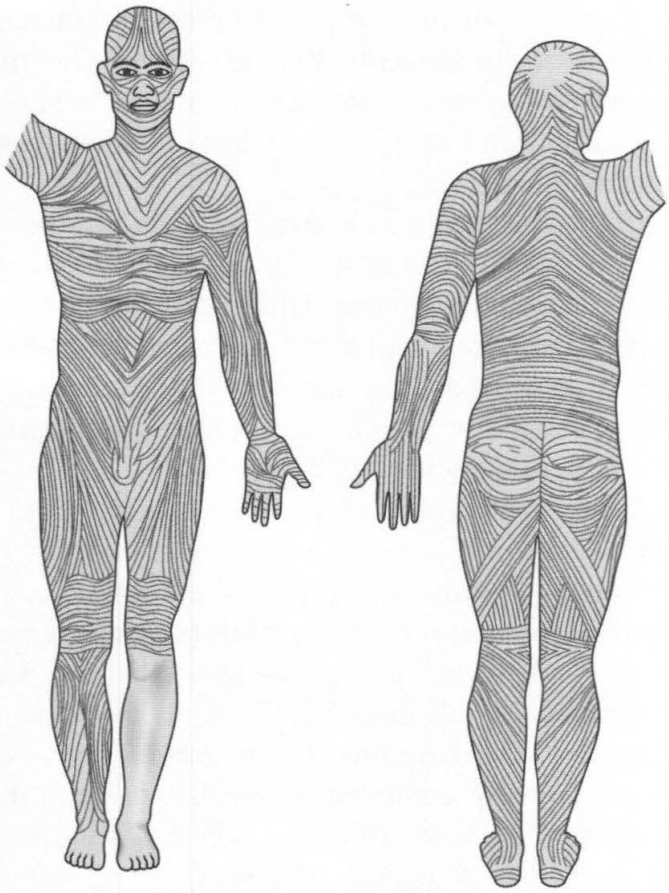


Fig. (8-11). Cleavage lines of Langer.

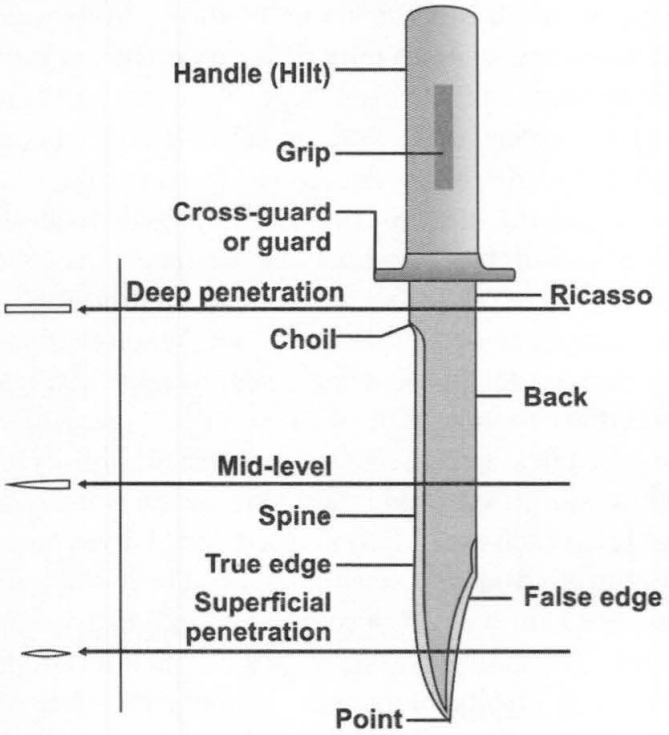


Fig. (8-12). Knife with clip-pointed blade. Differing skin wounds produced by same knife, at three different depths of penetration.

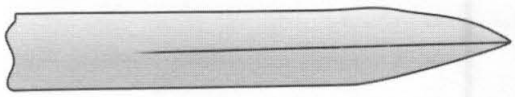


Fig. (8-13). Spear-pointed blade.

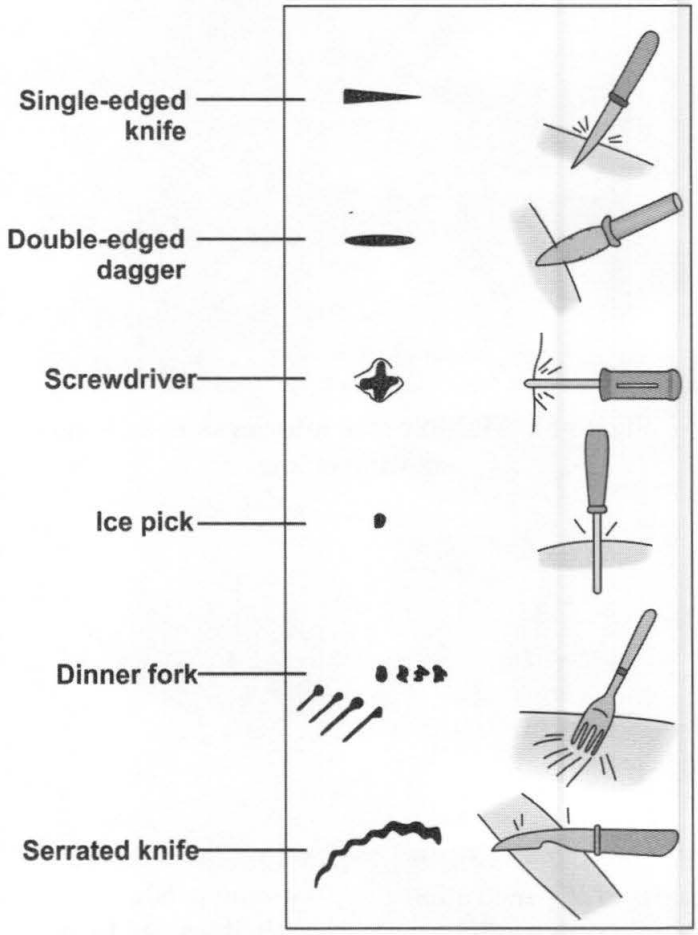


Fig. (8-14). Skin wounds produced by different weapons.

because of ricasso (short, unsharpened section of blade between the cutting edge and guard). When the full length of such knife up to the handle enters the body, both ends will be blunt. (b) If a double-edged weapon is used, the wound will be elliptical or slit-like and both angles will be sharp (**Figs 8-12 and 8-13**). If the knife penetrates to full length up to the guard, one or both edges may be blunt because of ricasso. (c) A round object like the spear may produce a circular wound. (d) A round blunt-pointed object, such as a pointed stick, or metal rod may produce a circular surface wound with inverted irregular and bruised edges. (e) A pointed square weapon may produce a cross-shaped injury, each of the four edges tearing its way through the tissues. (f) A fall on a pointed article,

e.g., pieces of broken glass will produce a wound with irregular and bruised margins, and fragments of glass may be found embedded in the soft tissues. (g) A screwdriver will produce a slit-like stab wound with squared ends and abraded margins. (h) When a knife is twisted as it is withdrawn from the tissues, the wound may be cruciate. (i) Ice-picks or similar instruments produce stab wounds resembling small calibre bullet wounds. (j) Irregularly-shaped stab wounds, such as L- or V-shaped are produced by stabbing, followed by simultaneous twisting and cutting (rocking), or the victim moving relative to the knife, or by a combination of the two. (k) The skin wound made by closed scissors produce a flat Z-shaped wound. (l) A knife with a serrated back edge will produce a stab wound, the back edge of which may be torn or ragged. (m) If the instrument is thrust in and is then completely withdrawn with the cutting edge dragging against one end, the wound would be extended superficially producing a tail. (n) If the instrument is thrust in and then pulled in the direction of a cutting edge, the track and the skin wound are elongated. (o) In a homicidal stab wound, the knife may be pulled upwards or downwards during insertion or withdrawal which cuts the skin. In such case, the wound is longer than the widest part of the blade. Accurate estimation of the blade can be made by bringing the margins into a straight line.

(6) Direction: When the knife penetrates at an angle, the wound will have a bevelled margin on one side with undermining, so that subcutaneous tissue is visible, indicating the direction from which the knife entered. In solid organs like the liver, the track made by the weapon is better seen. The principal direction should be noted first and other next, e.g., backwards and to the right. If the weapon is partially withdrawn and thrust again in a new direction, two or more punctures are seen in the soft parts with only one external wound. If the wound is perforating, it should be described in sequential order: the wound of entrance, the path of the wound track, and the wound of exit. If the wound is penetrating, the wound of entrance should be described first, then the depth and direction of wound track, and specific termination.

Complications: (1) External haemorrhage is slight, but there may be marked internal haemorrhage or injuries to internal organs (**Plate 9-51**). (2) The wound may get infected. (3) Air embolism may occur in a stab wound of the neck which penetrates jugular veins. (4) Pneumothorax. (5) Asphyxia due to inhalation of blood.

Concealed Puncture Wounds: These are puncture wounds caused on concealed parts of the body, such as nostrils, fontanelle, fornix of the upper eyelids, axilla, vagina, rectum, and nape of the neck. Fatal injuries can be caused without leaving any external marks, e.g., thrusting a needle or pin into the brain through the fontanelles, through the inner canthus of the eye, or into the medulla through the nape of the neck. These injuries may not be detected unless searched carefully.

Incised Stab Wound: It is a wound which starts as an incised wound and ends as a stab wound by the sudden thrust of the blade into the body, or starts as a stab wound and becomes incised wound as the knife is pulled out of the body at a shallow angle to the skin surface producing an incised wound (**Fig. 8-8**).

EXAMINATION: The following points should be noted: (1) Identification and labelling of cuts and damage to clothing. (2) Distribution of bloodstains. (3) Removal of clothing, layer by layer. (4) Identification and labelling of wounds. (5) Wounds: (a) position (height from heels), (b) location (measurement from fixed anatomical landmarks), (c) description including size, shape, extension, (d) direction, (e) depth, (f) trauma to viscera, (g) estimation of force required, (h) foreign bodies. The relationship between the wound and weapon can be established by the shape, width, length and presence of blood on the weapon. When multiple stab wounds are present, they should be numbered and photographed. A sketch or a printed body diagram should be used.

Postmortem stab wounds are usually yellow or tan in colour due to absence of tissue perfusion.

Medicolegal Importance: (1) The shape of the wound may indicate the type of the weapon which may have caused the injury. (2) The depth of the wound will indicate the force of penetration. (3) If a broken fragment of weapon is found, it will identify the weapon or will connect an accused person with the crime. (4) Direction and dimensions of the wound indicate the relative positions of the assailant and the victim. (5) The age of the injury can be determined. (6) Position, number and direction of wounds may indicate manner of production, i.e., suicide, accident, or homicide.

Table (8-5). Difference between incised, lacerated and stab wound.

	Trait	Incised wound	Lacerated wound	Stab wound
(1)	Manner of production:	By sharp objects or weapons.	By blunt objects or weapons.	By pointed, sharp or blunt weapons.
(2)	Site:	Anywhere.	Usually over bony prominences.	Anywhere; usually chest and abdomen.
(3)	Margins:	Smooth, even, clean-cut and everted.	Irregular and often one margin undermined.	Clean-cut; lacerated if weapon is blunt-pointed.
(4)	Abrasions on edges:	Absent.	Usually present.	Absent.
(5)	Bruising:	No adjacent bruising of soft tissues.	Bruising of surrounding and underlying tissues.	Rare.
(6)	Shape:	Linear or spindle-shaped.	Varies; usually irregular.	Varies, linear or irregular.
(7)	Dimensions:	Usually longer than deep; often gaping.	Usually longer than deep.	Depth greater than length and breadth.
(8)	Depth of wound:	Structures cleanly cut to the depth of wound.	Small strands of tissue at the bottom bridge across margins.	Structures cleanly cut.
(9)	Haemorrhage:	Usually profuse and external. Spurting may be seen.	Slight except scalp and external.	Varies; usually internal.
(10)	Hair bulbs:	Cleanly cut.	Crushed or torn.	Usually clean-cut.
(11)	Bones:	May be cut.	May be fractured.	May be punctured.
(12)	Foreign bodies:	Absent.	Usually present.	Usually absent.

Circumstances of Injuries

SUICIDE: Suicidal stab wounds are found over accessible areas of the body. The common site is the chest over the heart region. The depth is variable, some are superficial and others enter the pericardium or heart. Stab wounds of the head are rare. Usually, a number of stabs extending only to the bone are seen in the temporal region.

Common methods of suicide are: Poisoning, hanging, burning, drowning, jumping from a height, stabbing and cutting and railway injuries.

Hara-kiri: It is an unusual type of suicide, in which the victim inflicts a single large wound on the abdomen with a short sword while in a sitting position or falls forward upon a ceremonial sword and pulls out intestines. The sudden evisceration of internal organs causes a sudden decrease of intra-abdominal pressure and cardiac return, producing sudden cardiac collapse.

HOMICIDE: Most deaths from stab wounds are homicidal, especially if found in an inaccessible area, e.g. back of the body. In case of a sudden surprise attack, a single wound is found at a vital spot. If there is a struggle, there may be a number of wounds, sometimes associated with defence cuts on the hands. In OVERKILL HOMICIDE, assailant continues stabbing beyond the victim's death.

ACCIDENT: Accidental wounds are rare. They are caused by falling against projecting sharp objects like glass, nails, etc., or a person may be gored by the horns of a bull, buffalo, etc. (Tables 8-5 and 8-6).

DEFENCE WOUNDS: Defence wounds result due to the immediate and instinctive reaction of the victim to save himself. They are classified into two types: (1) **Active defence wounds** are caused when the victim tries to grasp the weapon. (2) **Passive defence wounds are caused when the victim raises the hands, arms or legs (Plate 11-64 and 11-65).** If the weapon is blunt, bruises and abrasions are produced on the extensor or ulnar surfaces of the forearm or back of the hands and knuckles. If the weapon is sharp, the injuries will depend upon the type of attack, whether stabbing or cutting. In stabbing with a single-edged weapon, if the weapon is grasped, a single cut is produced on the palm of the hand or the opposing bends of the fingers or thumb. If the weapon is double-edged, an opposing second cut is seen. The cuts are usually irregular and ragged, because the skin tension is loosened by gripping of the knife.

Table (8-6). Difference between suicidal, homicidal and accidental wounds.

	Trait	Suicide	Homicide	Accident
(1)	Nature of wounds:	Usually incised and stab.	Usually chop wounds, lacerations and stab.	Usually lacerations, abrasions and contusions.
(2)	Number of wounds:	Multiple.	Multiple.	Single, or multiple.
(3)	Target area:	Accessible part only, i.e., front and sides of the body, such as neck, wrists, left side of chest, groin, etc.	No fixed site; vital parts, such as head, chest, abdomen.	Anywhere; usually on exposed parts and bony prominences. Number of wounds on the same side.
(4)	Wound grouping:	Arranged.	Irregular.	Vulnerable parts.
(5)	Direction:	In right-handed persons from left to right and from above downwards.	Any direction.	Any direction.
(6)	Severity:	Mostly superficial; one or two deep wounds.	Mostly severe and extensive.	Variable severity.
(7)	Hesitation marks:	Usually present.	Absent.	Absent.
(8)	Defence wounds:	Absent.	May be present.	Absent.
(9)	Secondary injuries:	Absent.	May be connected with fight.	May be associated with accident.
(10)	Weapon:	By the side of the body or may be grasped firmly due to cadaveric spasm.	Absent.	Present.
(11)	Clothes:	Not damaged as they are usually removed.	May be damaged.	May be damaged and stained with oil, grease, mud, dirt.
(12)	Scene of crime:	Usually inside closed room; no disturbance of surroundings.	Disturbed and disorderly with signs of struggle and blood stains.	Varies with the nature of the accident.
(13)	Motive:	Present, such as domestic worries, disappointment in love. chronic disease, failure in examination, etc.	Revenge, robbery, sexual offences	Absent.

When attacked with a cutting weapon, the victim usually holds up the hand or forearm, and receives cuts on the hand, wrist, ulnar border of the forearm and on the fingers. They are often irregular in depth and distribution. Rarely, they may be found on the shins and feet if the victim was lying on the ground usually face up, as he kicks at the assailant, or curls up or tries to cover vital areas with his legs. Defence wounds also occur in firearm injuries, where an arm is raised in an attempt to shield the trunk or head. Defence wounds indicate homicide. **Defence wounds are absent,** if the victim is unconscious or is taken by surprise, or attacked from the back, or under the influence of alcohol or drugs.

Offensive Manual Injuries: Abrasions and contusions over the knuckles and sometimes fractures of fourth and fifth metacarpals can be sustained due to offensive efforts by the victim, or by his defensive efforts.

Therapeutic Wounds: They are the wounds produced during treatment, such as surgical incisions, venesection, tracheostomy, defibrillators, etc.

SELF-INFLICTED AND FABRICATED WOUNDS: Self-inflicted wounds are those inflicted by a person on his own body. **Fabricated wounds (fictitious, forged or invented wounds) are those which may be produced by a person on his own body or by another with his consent (self-suffered).**

Motive: They may be produced for the following reasons. (1) To charge an enemy with assault or attempted murder. (2) By women to bring a charge of rape against an enemy. (3) To make a simple injury appear serious. (4) By the assailant to pretend self-defence or to change the appearance of wounds which might connect him with the crime. (5) By policemen and watchmen acting in collusion with robbers to show that they were defending the property. (6) In thefts by servants or messengers for the above reason. (7) By recruits to escape military service.

Types of wounds: Fabricated wounds are mostly incised wounds, and sometimes contusions, stab wounds and burns. Lacerated wounds are rarely fabricated. Incised wounds are usually superficial, multiple, parallel and of equal depth at origin and termination. They avoid vital and sensitive areas like the eyes, lips, nose and ears. Some mentally disordered persons may inflict hundreds of small wounds upon themselves. Stab wounds are usually multiple and superficial and seen about the left arm or shoulder and sometimes on the chest. Burns are superficial and seen usually on the left upper arm. **The clothes are not cut and if cuts are seen they are not compatible with the nature of the wounds. The history of the assault is incompatible with the injuries.**

FORENSIC BALLISTICS

A **firearm** is any instrument which discharges a missile by the expansive force of the gases produced by combustion of an explosive substance. **Forensic ballistics is the science dealing with the investigation of firearms, ammunition and the problems arising from their use. Wound ballistics relates to the missiles effect on living tissue. Terminal ballistics** involves the study of behaviour of missiles once they penetrate their targets.

GENERAL MAKE-UP AND MECHANISM: Firearms consist of a metal barrel in the form of hollow cylinder of varying length, which is closed at the back end and is called the breech end; and the front open end is called the muzzle end. The inside of the barrel consists of three parts: (1) The chamber, at the breech end to accommodate the cartridge, (2) the taper, connects the chamber to the bore, and (3) the bore, which lies between the taper and the muzzle. When the weapon is closed for firing, the barrel comes into position against a flat block of metal called breech-face, which seals the breech end of the chamber. The block is pierced in its centre to accommodate the firing-pin, which moves forward when the trigger is pressed. The bolt or the block can be opened to insert a fresh cartridge into the chamber. A firearm is provided with sights with which one can aim, so that the bullet can strike the target accurately (Figs 8-15 and 8-17). The stock is the supporting or handle part of the weapon, the end of which is the butt. The handarms are provided with grips for grasping them by hand. In long-barrelled weapons, the butt is elongated to fit into the shoulder. Those with long barrels are fired from the shoulder, i.e., rifle and shotgun, and those with short barrels may be fired from the hand, i.e., pistol and revolver. Firearms have a safety device, which locks the firing mechanism when it is applied, known as safety catch.

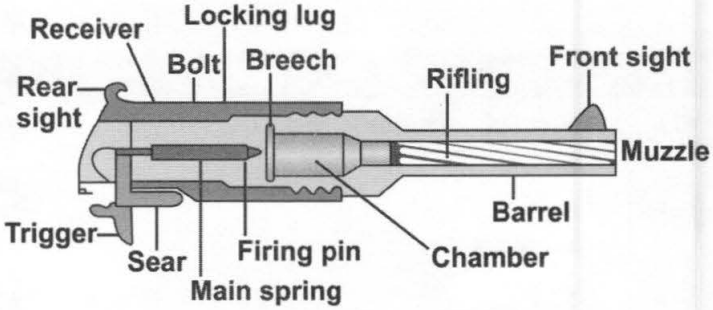


Fig. (8-15). Nomenclature of small arm.

Classification: (I) **Rifled weapons.** (1) Rifles: (a) Air and gas-operated rifles. (b) 0.22 rifles. (c) Military rifles. (2) Single-shot target practice pistols. (3) Revolvers. (4) Automatic pistols. (5) True automatic weapons (machine-guns).

(II) **Smooth-bored weapons** (shotgun): (1) Cylinder bore. (2) Choke bore. (3) Breech loader. (4) Muzzle loader. (5) Country-made.

(III) **According to barrel lengths:** (1) Side arms: They are guns with short barrel such as revolvers and pistols. (2) Shoulder arms: They are heavy long barrelled guns, which are fired from the shoulders such as shot guns or rifles.

(IV) **According to firing action:** (1) Over-bolt action. (2) Under-bolt action. (3) Lever action. (4) Pump-action or autoloading models.

CALIBRE OR GAUGE: It is measured by the internal dimension of the barrel and is given in decimals of inch or millimetres. **The dimension of**

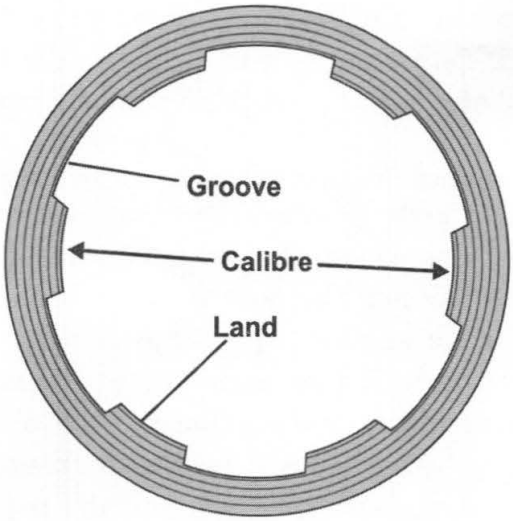


Fig. (8-16). Cross-section of a rifled barrel.

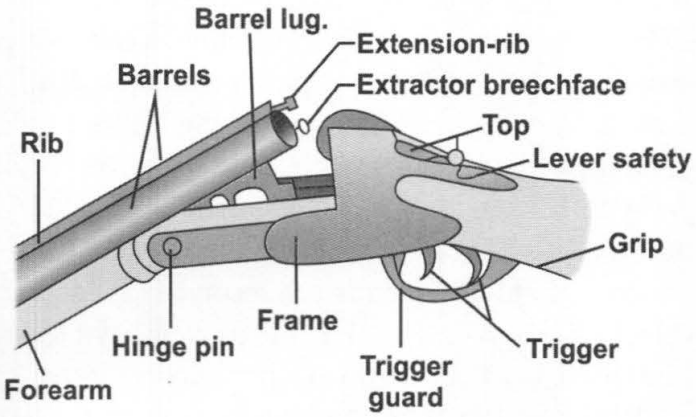


Fig. (8-17). Diagram showing details of twin-barrel (smooth-bore) shot gun.

the rifled weapon is measured between a pair of diametrically opposed lands and not grooves (Fig. 8-16). In smooth-bored weapons, the bore is measured similarly up to 1.27 cm. (half inch) (Fig. 8-16). For larger bores, the size is determined by the size of the lead ball which will exactly fit the barrel, and by the number of such balls of equal size and weight as can be made from 454 g (one pound) of pure lead. Thus the 12 bore gun is one, whose diameter is that of a ball of such a size that 12 may be made from 454 g. The smaller the number of gauge, the greater the diameter of the barrel.

Shotgun: It may be single-barrelled or double-barrelled. **It is intended for firing a single ball, slug or a charge of shots.** The barrel varies in length from 55 to 72 cm. **Shotgun bores vary from 4 to 20. The common gauges are 12, 16 and 20.** The weapon is made to break or open on hinge across the breech-facing, for the insertion and extraction of cartridge

Table (8-7). Various degrees of choke

	Designation	Constriction in mm.
(1)	**Full-choke**	**1.00**
(2)	**Three-quarter choke**	**0.75**
(3)	**Half-choke**	**0.50**
(4)	**Quarter-choke**	**0.25**
(5)	**True cylinder**	**0**

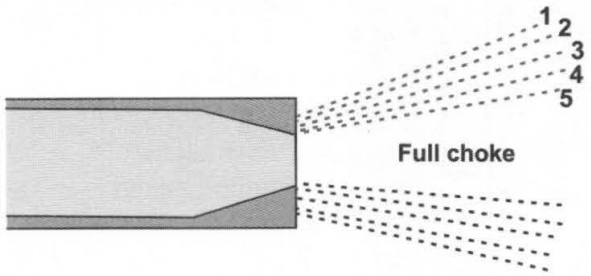


Fig. (8-18). Choking of shotgun: (1) Improvide cylinder, (2) Quarter choke, (3) Half choke, (4) 3/4 choke, (5) Full choke

cases. **The interior of the barrel is smooth. When the entire barrel from breech to muzzle is of the same diameter it is called cylinder-bore. In choke-bore, the distal seven to ten cm. of the barrel is narrow.** Different degrees are known as full-choke, half-choke and quarter-choke or improved cylinder **(Table 8-7 and Fig. 8-18)**. Shotguns may have variable choke-adapters. The **choking lessens the rate of spread of shot after it leaves the muzzle, increases the explosive force and increases the velocity.** There are some shotguns which have small portion of their bore near the muzzle end rifled, which are called **"paradox guns".** Some shotguns have the whole of their bore rifled with very shallow grooves. A **musket** is a military shoulder firearm. It has a long forestock and usually takes a bayonet (pointed knife-like weapon) at the muzzle. It is a smooth-bore weapon, e.g., 0.410 musket, which fires a single shot at high velocity, and is effective up to 90 metres. Shotguns are effective up to 30 metres. **Muzzle-loading guns** are loaded entirely from muzzle end with the help of a rod using gunpowder, pieces of cloth, stones, metal fragments, seeds, bolts, wood, screws, etc.

CARTRIDGE: The shotgun cartridge consists of a case of short metal cylinder which is continuous with a cardboard or plastic cylinder. The case is rimmed, which keeps the cartridge correctly in the chamber and facilitates extraction. The case helps

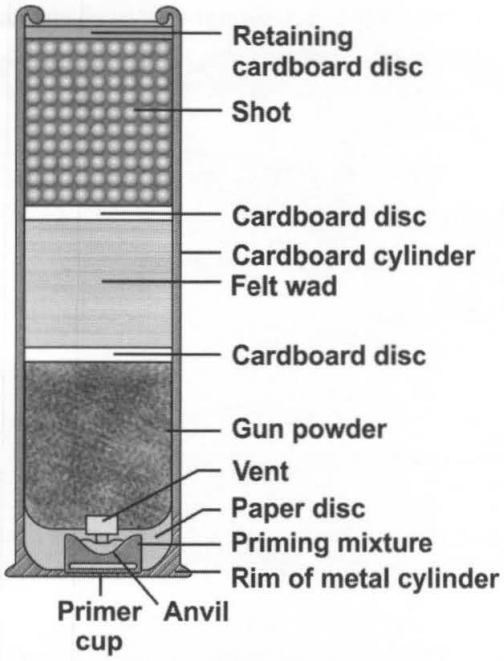


Fig. (8-19). Shotgun cartridge.

to keep the various components in place, prevents the backward escape of the gases, and provides a waterproof container for the gunpowder. The length of the cartridge varies from five to seven cm. **The cartridge case is filled as follows from the base: percussion cap (primer battery cup, detonator cap), gunpowder, a thick felt-wad with cardboard discs lying in front and behind it, the shot (charge or load), and finally the retaining cardboard disc,** over which the edges of the cartridge cylinder walls are pressed **(Fig. 8-19)**. Wads may be glazed-board, straw-board, plastic, cork, felt, etc., and may be disc-shaped, cup-shaped or bizarre-shaped. The diameter of the wadding used in the cartridge is greater than that of the bore of the gun. **Wad acts as a piston and seals the bore completely, thus preventing the expanding gases from escaping and disturbing the shot charge and allows optimum pressure to develop.** The felt wad contains grease, which lubricates the bore after the firing of each round. The powder is protected from the grease wad by a thin grease-proof card wad. The card wad behind the shot charge prevents the pellets from getting lodged in the felt wad. **Some cartridges contain "power piston" (Fig. 8-27),** which holds the shot inside a polythene cup formed from four leaflets or petals folded so as to produce a cylindrical shape, which may contribute to the wound at short range. **Rifled slugs** are single missile and are used in shotguns for big game hunting. They are similar in shape to a blunt bullet with a deep hollow cavity in base. The spiral grooves on the slugs impart a spinning effect **(Plate 9-54)**. The slugs have much greater range than pellets.

Improvised firearms or country-made firearms are common in India. They are of various calibres but 12 bore is the commonest. They are usually smooth-bored.

Rifled Arms: The bore is cut internally with a number of shallow, spiral "grooves", varying from two to more than twenty, the most common being six, which run parallel to each other but twisted spirally, from breech to muzzle. These **grooves are called "rifling", and the projecting ridges between these grooves are called "lands".** Riflings vary in number, direction, depth and width. When the bullet passes through the bore, its surface comes into contact with the projecting spirals which give the bullet a spinning or spiralling motion. Rifling is made by a broach**. Rifling gives the bullet a spin, greater power of penetration, a straight course and prevents it from unsteady movement as it travels in the air.** Rifled firearms are categorised into: (1) Low velocity (up to 360 metres per second). (2) Medium velocity (360 to 750 m/s). (3) High velocity (750 to 1260 m/s). (4) Very high velocity (above 1260 m/s).

RIFLE: A rifle is a gun with a long barrel, the bore of which is rifled. A CARBINE is a short-barrelled rifle, or a musket. It is effective up to 300 metres. The military rifle has a magazine and bolt action, muzzle velocity of 800 metres per second and can kill at a range of 3,000 metres. Rifles may be single-shot, repeating, semi-automatic and automatic (Fig. 8-20).

REVOLVER: Revolvers are so-called because the ammunition is put in chambers in a metal cylinder which revolves or rotates before each shot, to bring the next cartridge opposite the barrel, ready to be fired. It has a cylindrical magazine situated at the back of the barrel, which is capable of revolving motion, and can accommodate 5 to 6 cartridges, each housed in a separate chamber. The bores vary from 5.6 to 11.4 mm. (0.22 to 0.45 inch). They are low velocity weapons. The effective range is hundred metres (Fig. 8-21).

AUTOMATIC PISTOL: In this, when a cartridge is fired, the empty cartridge case is thrown out and a new cartridge slips into the breech automatically by a spring. The cartridges are contained in a vertical magazine in the stock, which can accommodate six to ten cartridges. They are really semi-automatic or self-loading because the trigger has to be pressed each time a round is fired.

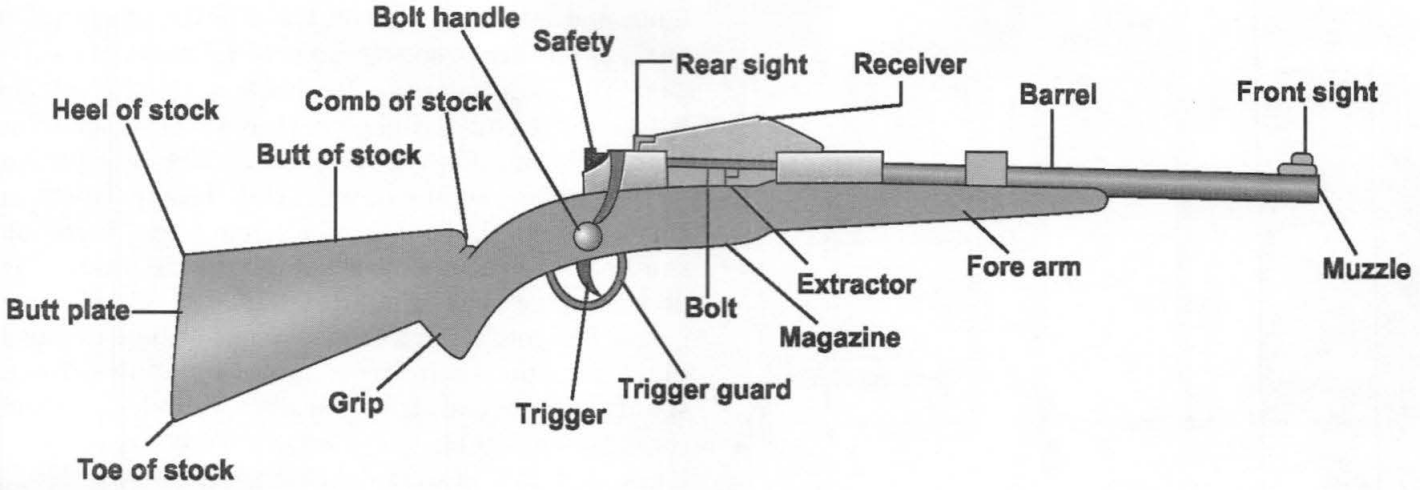


Fig. (8-20). Diagram showing general characters of a rifle.

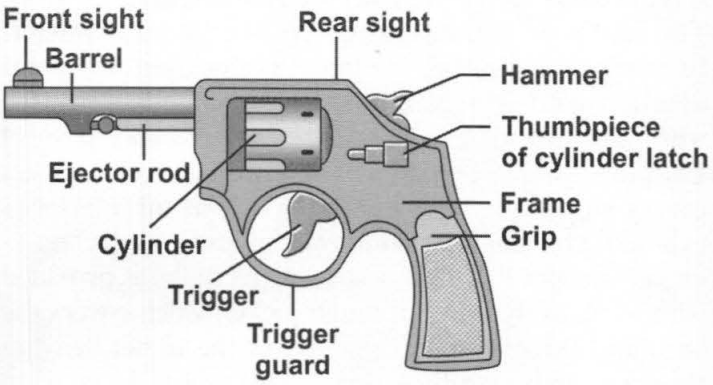


Fig. (8-21). Diagram showing general characters of a revolver.

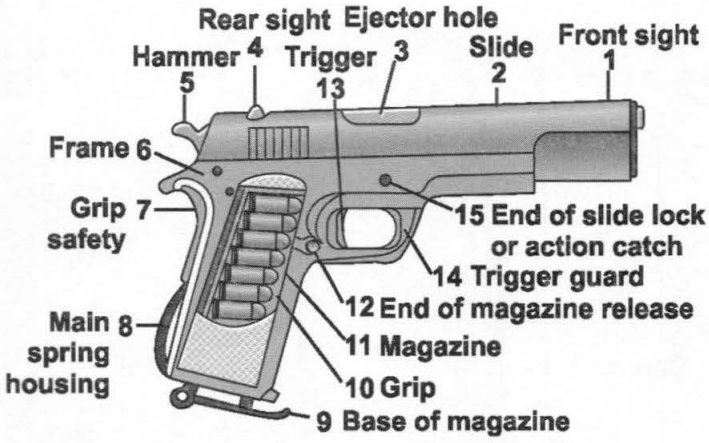


Fig. (8-22). Diagram showing general characters of a pistol.

The bores vary from 6.35 to 11.4 mm. (0.25 to 0.45 inch). They are high velocity weapons. Their effective range is about hundred metres (Fig. 8-22).

AIR RIFLE AND AIR PISTOL: In these, compressed air is used to fire lead slugs. Some weapons use cartridges of liquid CO_2 as the propellant. The velocity is very little and usually minor injuries are caused. Death can occur from injury to the heart or large blood vessels.

Small arms include hand arms and shoulder arms.

AUTOMATIC WEAPONS: Machine-guns or sub-machine guns continue to fire through the action of gases of the previous explosion and of the ejector and recoil spring.

CARTRIDGE: It consists of a metal cylinder with a flat base which projects as a rim, except in an automatic pistol. Rimless cartridge has an extractor groove near the base. **The primer cup (percussion cap) is fitted in a circular hole, usually in the centre of the base. The metal cylinder or cartridge case is elongated and its distal end tightly grips the base of the projectile or bullet.** The gunpowder lies between the detonator and the bullet. Usually there is no wad, but sometimes one piece wad is kept. Lower power rim-fire cartridge may not contain gunpowder, but only a primer compound in a hollow circumferential rim. Many bullets have, near the base, a circumferential groove called **"cannelure"**, into which the end of the case is crimped. A bullet without cannelure is held in position by stabs on the circumference of the case **(Figs 8-23 and 8-24)**.

Primers: Centrefire rifle and pistol primers are small metal cups containing the priming mixture and an anvil. The blow of the firing pin on the primer cup crushes the priming mixture against the anvil centre and burns it, which then flashes through the flash-holes (fire-holes or vents) and burns the powder. The primer used in shotgun cartridge is called a primer battery cup. This consists of a battery cup into which a

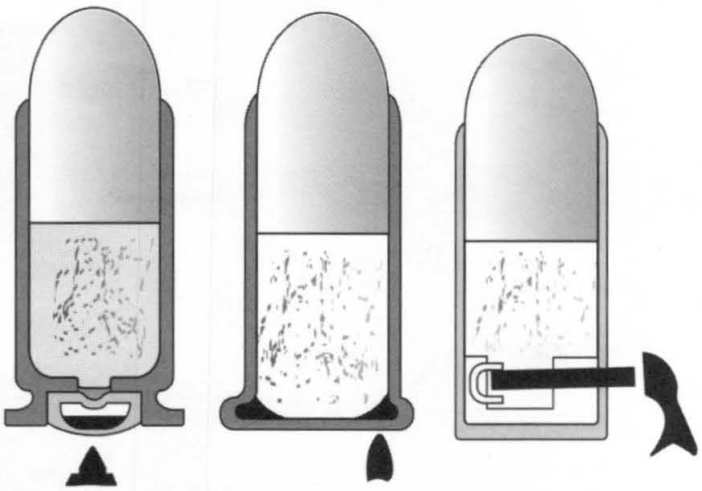


Fig. (8-23). Three types of cartridges.
(1) Centerfire. (2) Rimfire. (3) Pin-fire.

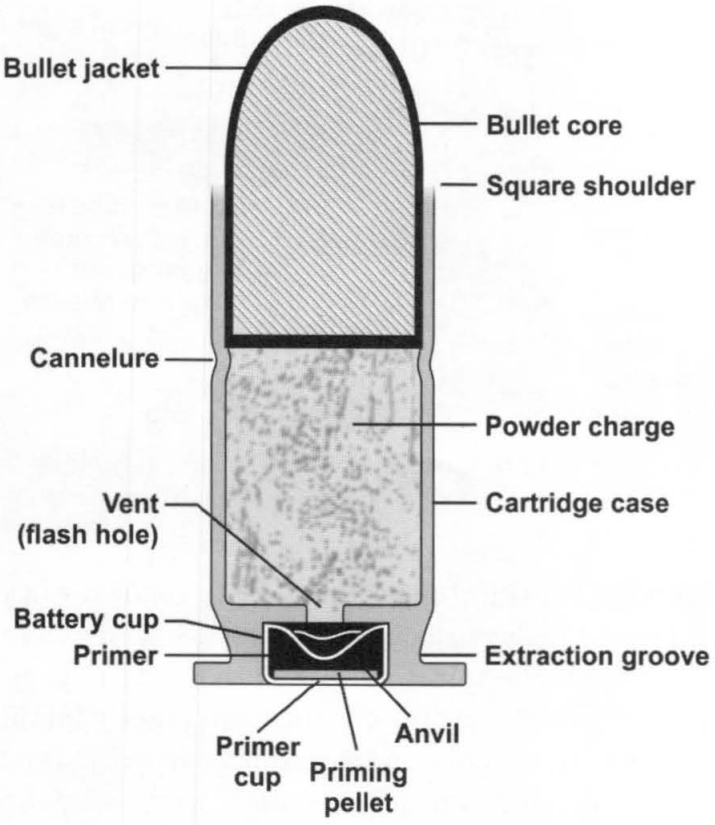


Fig. (8-24). Cartridge of rifled weapon.

primer cup fits. The battery cup supports the anvil and provides flash holes. The priming mixture contains lead peroxide, lead styphnate, antimony sulphide, tetrazene, barium nitrate, etc.

POWDERS: **(1) Black Powder: It consists of potassium nitrate 75%; sulphur 10%; and charcoal 15%. It is designated as FG, FFG, FFFG, etc., depending on the size of the grains. The more number of F's, the finer are the grains and the faster in burning. The powder grains are black, coarse or fine without any particular shape. It burns with production of much heat, flame and smoke. Fine grains travel 60 cm. or more. One gram of powder produces 3,000 to 4,500 c.c. of gas.**

(2) Smokeless Powder: It consists of: (1) Nitrocellulose (single base). (2) Nitroglycerine and nitrocellulose (double base). (3) Nitrocellulose, nitroglycerine and nitroguanidine (triple base). Triple base powders are commonly used. They produce much less flame and smoke and are more completely burnt than black powder. One gram produces 12,000 to 13,000 c.c. of gases. The colour varies from bright-orange to bluish-black and the shape from minute globules, flakes, square, rectangular, irregular discs, cylinders to longer threads. Semi-smokeless powder is a mixture of 80% of black, and 20% of the smokeless type.

BULLETS: **The traditional bullet is made of soft metal and has a rounded nose. The metal is lead with varying amounts of antimony added to provide hardness. The missile is usually known as the round-nose soft bullet. It is the bullet most commonly used in rifles and revolvers. The calibre of a bullet is its cross-sectional diameter. In revolver and pistol, the bullet is short and the point usually round or ogival. In rifle, the bullet is elongated with pointed end. Jacketed bullets are of two types: (1) The full metal jacket bullet in which a tough, heavy jacket covers all but the base where the soft metal interior is exposed. The tough jacket may be made of steel, copper, nickel and zinc. (2) The semi-jacketed bullet is provided with a relatively thin but tough jacket which covers the base and the cylindrical portion of the bullet, leaving the nose partly or fully exposed. This type is designed to expand or "mushroom" like the soft metal, hollow-point type. A dumdum bullet is one which fragments extensively upon striking (out lawed). Frangible bullet is made of iron which fragments (just-like particles) on impact. Incendiary bullets contain phosphorus, so that it catches fire on hitting the target. Tracer bullet leaves a visible mark or "trace' while in flight, due to which the gunner can observe the strike of the shot (Fig. 8-25).**

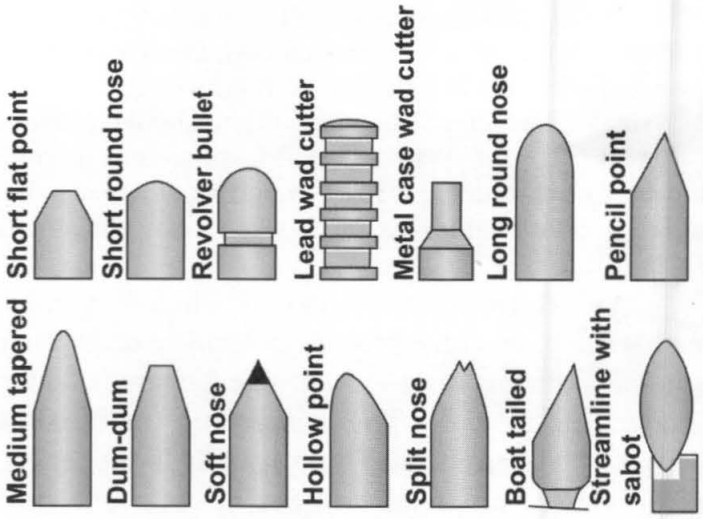


Fig. (8-25). Types of bullets.

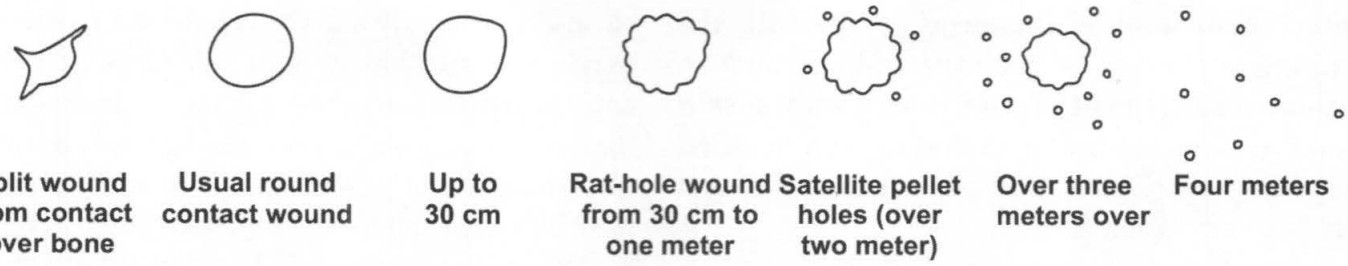

Split wound from contact over bone
Usual round contact wound
Up to 30 cm
Rat-hole wound from 30 cm to one meter
Satellite pellet holes (over two meter)
Over three meters over
Four meters

Fig. (8-26). Shotgun wounds at varying distances.

MECHANISM OF DISCHARGE OF PROJECTILE: A firearm is fired when the trigger is pulled, which is usually situated below, or below and behind the breech. The trigger releases a pin or hammer whose tip strikes the percussion cap at the base of the cartridge, which contains explosive mixture known as priming, which explodes by heat created by the strike of the firing pin and sends a flash through a tiny hole into the main body of the powder-filled case, which sets fire to the powder charge, producing a large amount of gas, which is under high pressure. The cartridge case swells outwards due to which the hold on the bullet is released, and forces the bullet into the barrel. The projectile reaches its maximum velocity as it comes out at the open end of the barrel, and this is called the muzzle velocity. The bullet is followed by a blast of highly compressed hot gas, particles of partially burnt and unburnt powder, smoke, flame and fragments of metal and grease. The blast assumes the shape of a cone, whose apex is located at the muzzle. All these things produce some effect on the body at short ranges.

FIREARM WOUNDS

WOUNDS FROM SHOTGUNS: Shotgun discharge contains lead pellets, soot in the form of smoke and debris, unburnt and burning propellant flakes or grains, flame and hot gases under pressure, detonator constituents and fragments of cartridge case. The smoke extends up to 30 cm., flame up to 45 cm., and unburnt and partially burnt powder grains up to 60 to 90 cm. The cards travel for two metres, and wad for 2 to 5 metres. Marked variations occur between different weapons, different barrels of the same weapon, and different ammunitions fired from the same weapon. **Ammunition plays a great role in the size and pattern of wounding**.

The character of a wound depends on **(I) The Distance from which the Weapon is Discharged:** The discharge produces a long, shallow cone with its apex close to the muzzle of the shotgun, and the farther along the cone the victim is situated, the larger the wound pattern will be **(Fig. 8-26)**.

(1) Contact and near contact wounds: The discharge from the muzzle, i.e., hot gases, smoke, flame, powder, cards, wad and metallic particles are blown into the body. **If the contact is tight, muzzle impression (copy or recoil abrasion) is seen (Plate 9-53).** Many muzzle impressions are not recorded due to the rapid removal of the weapon by recoil. When a firearm is fired, the shooter gets a jerk on the shoulder or wrist due to recoil movement of firearm. **In contact shot, the muzzle blast and the negative pressure in the barrel following discharge may suck blood, hair, fragments of tissues and cloth fibres several cm., back inside the barrel called "back spatter". The wound is single, usually round, equal to the bore in size, often ragged because of tearing due to gases.** In **tight contact**, wound soiling and burning are minimal or absent. **If the muzzle is not pressed firmly,** and also due to the recoil of the gun, flame, gas, and soot escape sideways and cause singeing and blackening of the surrounding skin. The margins of the skin perforation are charred by flame, and the abraded border is usually soiled with powder residue and smoke soiling of at least some of the margin of the wound. As the gases are blasted within the wound, the subcutaneous and deeper tissues show severe disruption. Particles of unburnt powder are driven to some distance through the wound, and some of them are found embedded in the wound. These particles cause haemorrhage in deeper tissues and in the margins of the wound. Thus, the margins of the wound will be contused. CO in the gases combines with haemoglobin due to which the wound of entry and the wound track appear pink. Burning blackening and tattooing of the tissue also take place in the depths of the wound. **In loose contact or near**

contact shot, some of the gases escape with the resulting scattering of the muzzle blast and an unusual arrangement of soot is seen on the skin known as corona, similar to that as seen in rifled weapon wound. The internal damage is diffuse, but there is no cavitation.

Contact shot over a bony area: Cruciate, stellate, or ragged lacerations are seen especially if there is a thick bone immediately under the skin. **Contact wounds on the head** produce marked disruption of the margins, and often show subsidiary linear tears in the skin, extending from the margins of the main wound. A large irregular hole is produced in the skull, with fissured fractures running from its margins **(burst head)**. The bone may show burning and blackening in the margins. Extreme mutilation is caused due to the explosive effect, because the gases have restricted space for expansion. The entire contour of the face and head may be destroyed, and the actual point of the muzzle impact of entry wound may be difficult to locate. Comminution of the vault of the skull is usually accompanied by extensive fissured fractures of the base of the skull, and the roofs of the orbits and middle ears. An eye may be blown out of its socket. A large exit wound may be produced with disruption of the cranium and projection of brain tissue for some distance. An entrance wound in the mouth or nose may or may not be accompanied by an exit wound. Resistance by the hard palate reduces the power of the shot, and the shots lie mostly in the space between the skull and scalp, at the back or side of the head. Splitting of the angle of mouth may occur due to blast.

In the neck region, a large exit wound or complete destruction may occur. **In contact or near contact wounds on the abdomen, coils of small intestine may lie outside the abdomen** due to entry of gas into the peritoneal cavity.

If the part is clothed, smoke will escape sideways and may be found in each layer of clothing and on the skin. The cloth may be singed at the edge of the hole, and there may be a ring of burning around the skin wound.

(2) **Close Range (up to one metre): The wound is single, circular or oval similar to contact wound, though the blackening and tattooing are more extensive.** The margins of the skin wound may be clean-cut or slightly ragged. **The deeper tissues show marked disruption.** Within a distance of about 30 cm., the tissues surrounding the wound are singed by flame and blackened by smoke and tattooed by unburnt or partially burnt powder granules. If the powder is smokeless, there may be greyish or white deposit on the skin round the wound. **The deposit of smoke is known as smudging, fouling or blackening.** This spreads more widely than powder tattooing. The blackening can be removed by a wet cloth. **Unburnt particles of the powder are embedded in the skin, producing tattooing (stippling or peppering).** The particles sticking to the epidermis or lodging in the epidermis superficially can be removed by wiping, showing punctate wounds. The hairs of the trunk and limbs may be completely burnt around the wound. **If the gun is fired at right angle to the body, the burnt area is circular, and if fired at an angle it is oval.** The end nearer the wound is the direction towards which the shot travelled. The burnt area darkens and parchmentises on drying after death. **The tissues up to 30 cm along the track and around the wound may be cherry-red due to absorption of CO.** As range becomes greater, the intensity of blackening and tattooing decreases and the spread increases in a fairly regular manner.

Between 30 cm. to one metre, the rim of the wound is irregular and shows some scalloping, often referred to as a **"rat hole"**. In some cartridges, plastic granules may be used as a filler between the lead pellets, and this coloured material may be found within the wound or on the skin, up to 2 to 3 metres and produce very fine, punctate abrasions around the pellet holes. **The plastic cup type wad opens up between 30 to 60 cm. so that four petals stick out, and a circular entrance wound is produced with a square-edged star or capital 'X' shape (Maltes cross) producing a characteristic bruise of abrasion of a similar shape encircling it (Fig. 8-27).** By 90 cm. air resistance folds back the petals and a single hole of entrance will be produced. Felt, wads or plastic cups from the cartridge will be found in the depths of the wound. **At a distance of 60 to 90 cm.** small shot produce a single, circular aperture three to four centimetres in diameter with irregular and lacerated edges. There is no burning or blackening. Some amount of tattooing is usually seen. The tissues within and around the wound are not cherry-red. The

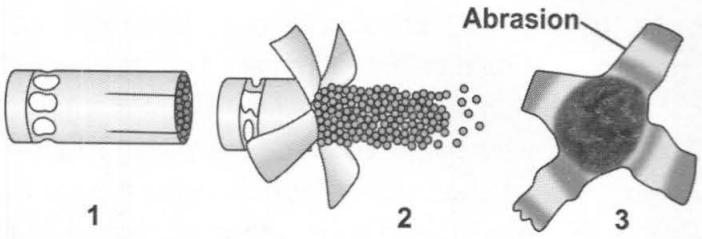


Fig. (8-27). Shotgun wad with enclosed lead shot in plastic cup. (1) Cup is not opened. (2) Between 30 to 60 cm. the sleeves of cup open and are forced backward. (3) Cross-like pattern of abrasion around the entry wound.

shots are scattered after entering the body and cause much damage to the internal tissues. In the **skull**, there is less destruction than that at contact range; bursting open of the skull and scattering of its contents is not seen. Shotgun wounds at contact and close range cause much more destruction of tissues than rifled weapons, due to the greater amount of gas produced because of larger amount of powder.

(3) Short range (1 to 2 metres): Between one and two metres, the shot mass enters the body in one mass, producing a round hole, four to five cm. in diameter, with irregular and lacerated edges. The margins are abraded. There is no burning, blackening or tattooing. Wad may be found deep inside the wound. Annular or linear abrasions are caused by the impact of the clothing against the stretched skin during the penetration of the shot *en masse.*

(4) Intermediate Range (2 to 4 metres): The minimal distance at which shot mass begins to spread is extremely variable. **At a distance of two metres, the shot mass begins to spread and individual pellet holes may be detected, which are usually round and show a rim of abrasion at their margins.** The wound of entry is irregular. Beyond two metres, the wads often strike the body below the shotgun wound. It may penetrate the skin or it may only bruise or abrade the skin. At a distance of three metres the central aperture is surrounded by separate openings in an area of about 8 to 10 cm. in diameter. **As muzzle-target distance increases, the main entrance defect progressively becomes smaller, and individual pellet wounds increase in number.** The outer shots are deformed by their passage through the bore and choke.

(5) Long or Distant Range (above four metres): Distant shots are those in which all shots penetrate separately. At a distance of four metres, the shots spread widely and enter the body as individual pellets producing separate openings in an area of ten to fifteen cm. in diameter (Plate 9-52 and 9-54). In shotgun discharge, there is no temporary cavitation. **The spread of pellets from a fully choked barrel is about (Fig. 826):**

Ten metres	-------	25 cm.
Fifteen metres	-------	35 cm.
Twenty metres	-------	45 cm.
Thirty metres	-------	75 cm.

The spread is almost double from an unchoked barrel. At about 30 metres, the pellets only penetrate the skin or muscle. The diameter of the wound is measured from the outermost of the individual pellet holes in centimetres. For cylindrical barrels, one-third of the spread of shot in cm. equals the range in metres. Death beyond a range of thirty metres is rare. If shotgun pellets pass through any target before they strike the body, the pellets spread, e.g., window glass, screen, or layers of clothing. Marked variations occur between different weapons and ammunition fired from the same gun.

(2) The Size of the Shot: The smaller the shot, the more minutely irregular are the edges. The penetrating power of the large shot is greater than that of the smaller shot. At close range, the wounds produced by shot of various sizes are similar, but small shot usually lodges in the body.

(3) The Nature of the Explosive: The extent of tattooing depends on the bore of weapon, the type of powder used, the weapon and range. The greater the bore of weapon, the wider the area of blackening. With smokeless powder, blackening and tattooing is less marked at all ranges than with black powder. Ammunition plays a great role in the size and pattern of wounding. Different brands of ammunition even when loaded with same shot size, produce different patterns at the same range.

(4) The Gun itself: The pattern of wound depends on the length of the barrel, the bore, absence or degree of the choke present. Shorter barrels usually produce greater deposits over larger areas. Silencers will filter out a great proportion of soot and powder particles, making the range appear greater than it actually was.

Irregular missiles, such as pieces of stone, iron, beads of metal, seeds, etc., used in muzzle-loading guns, produce several irregular lacerated wounds.

The direction of the fire may be determined from the passage of individual shots through the clothes and tissues, from the grouping of shot marks and the direction of glancing shots. Death from shotgun injury is usually due to haemorrhage. It is possible for a single pellet or shot to cause death.

Exit Wounds: Usually shotgun pellets do not exit from the body except: (a) Contact wounds. (b) Tangential wounds where some of the pellets have a very short track through the body. (c) Thin part of the body, such as the neck or extremities. (d) Wounds caused by large calibre buckshot or rifled slugs. **At contact or near range, greater disruption of tissues occur than is seen in entrance wound.** The margins are everted as the unsupported skin is struck from within, the tissues tend to burst outward, but there is no singeing, blackening, or tattooing of the margins. There may be small, separate wounds made by individual pellets that have become separated from the mass. Usually, a bruise may be seen in the deep tissue of exit site and shot may be felt under the skin.

WOUNDS FROM REVOLVERS AND AUTOMATIC PISTOLS

Entrance wound (in-shot wounds): They may be classified on the distance of the muzzle of a firearm from the body; contact shot, close shot, near shot and distant shot. **The flame extends up to eight cm.; smoke up to 15 cm.; and unburnt and partially burnt powder grains and small metallic particles up to 40 to 50 cm with handguns and up to 60 to 100 cm. in case of rifle.**

(1) Contact Shot: In firm contact, the resulting wound is similar to that from a shotgun. In some contact wounds, the imprint of the muzzle of the gun is found as patterned abrasion on the skin around the wound **(copy or recoil abrasion)**. Contact and close range discharges may cause pieces of skin, hair and adipose tissue fragments to enter the barrel for several centimetres called **'back spatter'** due to a momentary suction effect after pressure of gas blast subsides due to negative pressure in the barrel and possibly due to rapid relative cooling of the barrel. **Back spatter is more common with shotgun.** The discharge from the muzzle, i.e., gases, flame, powder, smoke and metallic particles are blown into the track taken by the bullet through the body. The wound is large, triangular, stellate, cruciate or elliptic, and shows cavitation due to the expansion of the liberated gases in the skin and tissues, which show laceration. The margins are contused and everted due to gases coming out of the entering wound under pressure **(Plate 10-55)**. The area immediately around the perforation is abraded, and this thin rim of abrasion is usually covered with powder residue. The margins of the wound of entry and the subcutaneous tissues may show pinkish-red discolouration due to presence of CO. The powder residue is usually grossly visible in the subcutaneous and deeper areas. The entrance track is blackened and burnt. **Abdominal wounds** show cavitation because of the blast effect.

Head wounds show, as very large explosive type of injury with bursting fractures. The skin wound is large and irregular because of the expansion of gases between the scalp and the skull which causes eversion and splitting of the skin at the margins of the entrance wound. This results in **undermined, ragged, cruciform opening with everted margins.** Such wounds are usually produced by large calibered pistols. A subcutaneous pocket containing blood mixed with gunpowder is formed by separation of the tissues. Soot may be deposited on the bone surrounding the bullet hole. Frequently, soot passes deeper into the wound track, and a faint gray or black discolouration may be seen on the inner surfaces of the skull around bullet hole and on the dura mater. **Fissured fractures often radiate from the circular defect** due to the considerable sudden expansion resulting from the muzzle blast. Fractures of the orbital roofs occur due to the same mechanism. A bullet travelling the cranial cavity, destroys the structures in its pathway, and produces an expansile or explosive effect. When the bullet moves through the body, it transfers kinetic energy to the surrounding tissue, which is thrown forcefully away from the bullets path in a radial manner, and a temporary cavity is formed in its path. In less severe type of injury, a cone of damaged cerebral tissue is seen surrounding the wound track.

In **loose or near-contact shot,** some of the gases escape with the resulting scattering of the muzzle blast and an unusual arrangement of soot is seen on the skin known as **corona**. **The corona consists of a circular zone of soot deposit surrounding the bullet defect,**

but separated from it by a band of skin without a deposit of soot. This is due to the gas expanding about the muzzle, first at a velocity too high to allow for the settling out of soot, with a subsequent loss in velocity at a short distance from the muzzle, allowing the soot to finally deposit on the skin. The blast effect is not as marked as in tight contact and splitting of the wound edges does not occur. Singeing of the hair may be seen.

(2) Close Shot: This term is applied when the victim is within the range of the flame, i.e., up to 8 cm. The term 'point blank' is used when the range is very close to or in contact with the surface of the skin. The entrance wound is circular with inverted edges and is surrounded by blackened, tattooed and burnt area. A circular wound is produced only when the cone is at 90 degrees to the plane. In all other positions an ellipse wound is seen, its elongation increasing as the angle decreases. This pattern also applies to soot deposition (Plate 10-56). The length of the barrel of a firearm has considerable effect on the spread of smoke produced on the target, e.g., a gun with a five cm. barrel will spread the smoke over a much larger area than a weapon having a 15 cm. barrel, fired from the same distance and using the same type of ammunition. Soot is dark in the centre and fades towards the periphery. The wound track may be pinkish due to CO. Hair in the surrounding area may be clubbed, swollen at intervals by heat, or burnt. The blackening can be wiped off the skin. Unburnt and partially burnt powder grains will be driven into the skin producing tattooing. This cannot be wiped off the skin. Abraded collar and grease or dirt collar are present. The internal injuries are similar to the contact shot.

(3) Near Shot: This term is applied when the victim is within the range of powder blast and outside the range of flame, i.e., 10 to 50 cm. If the discharge occurs at a distance of about fifteen cm., the lacerating and burning effects of the gases are usually lost due to the dispersion cooling of the gases before they reach the skin. **The entrance wound is seen as a round hole; slightly smaller than the diameter of the bullet due to elasticity of the skin, with a bruised margin, and a zone of blackening and tattooing.** If the bullet strikes the body at an angle, blackening has a pear-shaped area, with the larger area on the side nearer the barrel. The individual tattoos are caused by the individual unburned powder grains being blown into the skin. A small magenta-coloured zone, an actual micro-contusion is seen surrounding each tattoo point, which is caused by the trauma of the high speed impaction of the powder grain with rupture of small blood vessels resulting in tiny haemorrhages. The blackening and tattooing is spread out over a large area, but there is no singeing of the skin (Plates 10-57 and 10-58). The size and density of powder tattooing pattern around wound of entrance vary with the calibre of weapon, length of barrel, type of propellant and the distance from the muzzle to the target. **The tissues up to 30 cm. along the track and around the wound may be cherry-red due to absorption of CO.** Between 30 cm. to 50 cm. there is no burning and blackening, but some amount of tattooing is usually seen. Abraded collar and grease or dirt collar are present.

(4) Distant Shot: Above one metre, the entrance wound is smaller than the bullet due to the elasticity of the skin, circular, and margins are inverted (Plate 10-59). Burning, blackening and tattooing are not seen. The skin adjacent to the hole shows two zones, the inner of grease collar and the outer of abraded collar. **In case of semi-jacketed bullet, the jacket separates as it goes through the body and the core mushrooms into small pieces. X-ray gives a picture of lead "snowstorm".** If a semi-jacketed bullet passes through an intermediary target, the jacket may separate from the core, and both missiles may penetrate the body. Superficial lacerations may be produced around the entry wound (pseudo-tattooing) which are larger and more irregular than that caused by powder.

THE ABRASION COLLAR (marginal abrasion): As the bullet strikes the skin, it first indents and then stretches the skin surface, so that perforation takes place through a tense area. After the bullet has perforated the skin, the elasticity of the skin causes the defect to contract. **The skin is abraded (abrasion collar) around the hole due to rubbing of the gyrating body of the bullet against the inverted epidermis.** Abrasion collar is reddish at first, but becomes brown and brownish-black as it dries (Plate 10-60). A black ring **"grease or dirt collar"** (bullet wipe), is seen as a narrow ring of skin, lining the defect

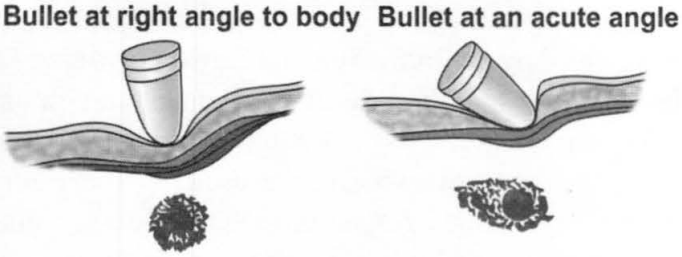


Fig. (8-28). Abrasion collar of an entrance bullet wound.

and is sharply outlined. This results from the removal of substances from the bullet as it passes through the skin, i.e., bullet lubrication, gun oil from the interior of the barrel, lead from the surface of the bullet, barrel debris, etc. **The abrasion collar surrounds the dirt collar. Some contusion is present in abraded collar, and as such, it is also called "contusion collar".** The abrasion and contusion collar is one to three millimetres wide (Fig. 8-28). These two features are proof of an entrance wound. Abrasion collar may be absent when the tissues are soft and yielding, e.g. in the abdomen or buttocks. In addition to abrasion collar, there is often a slightly wider circle of peeled keratin, where the stratum corneum of the skin is raised to form a slightly frayed edge around the entry wound.

Puppe's Rule: This rule is applicable to any multiple blunt force causing skull fractures. The test depends on the observations of the fracture lines either when they intersect each other or when they intersect a cratered lesion, so that one can determine which crack or defect must have been formed first.

Direction: **If the bullet strikes the body at right angle, the abrasion collar is circular and uniform,** because the scraping by the bullet is equal on all sides of the wound. **If the bullet strikes the body at an angle, the wound itself is round, but the marginal abrasion is oval due to increased width on one side, the actual width depending on the angle at which the bullet strikes the body. The abrasion and contusion collar is wider on the side from which the bullet comes, due to the bullet moving across a wider surface of the skin on that side.** The depth of the wound may be undercut, giving an indication of direction.

Skull: In the skull, the **wound of entrance shows a punched-in (clean) hole in the outer table. The inner table is unsupported and a cone-shaped piece of bone is detached forming a crater** that is larger than the hole on the outer table, and **shows bevelling** (sloping surface). Fissured fractures often radiate from the defects. As the bone fragments have to pass through the dura before entering the brain, lacerations are usually irregular and involve leptomeninges. Pieces of bone from wound of entrance are often driven into the cranial cavity and may establish the bullet track. Pieces of bone may produce short accessory wound tracks. **At the point of exit, a punched-out opening is produced in the inner table and bevelled opening on the outer table.** The exit wound is larger due to the deformity and tumbling of the bullet after entering the skull. Asymmetry of the bevelling is useful in assessing the angle of fire (Figs 8-29 and 8-30). The same appearance is seen in sternum, pelvis, ribs, dentures and thumbnails.

Airgun injuries: The missile is single and fired through a rifled barrel, 0.177 and 0.22 inch calibres are common. The pellet can enter skull and traverse whole width of brain but never produce an exit wound. The entry wound is small, but may show abrasion collar. There is no burning, blackening or tattooing.

Clothes: In a contact shot, the clothing usually shows a cross-shaped perforation and the skin around the bullet hole and the deeper layers of the cloth are blackened. In close shots, the clothing may absorb or filter out all of the products of discharge except the bullet. Sometimes, pieces of cloth are carried into the wound of entrance.

(B) Exit Wounds: Exit (outshoot) wounds may vary considerably in size and shape. They may be round, stellate, cruciate, elliptical, crescent-shaped, or appear as linear lacerations or even incised wounds. Exit wounds of the head are usually star-

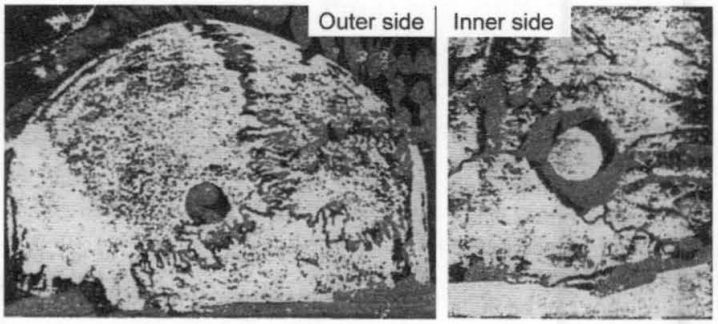


Fig. (8-29). (A) Skull cap showing the entrance hole of a revolver bullet with clean-cut margins. (B) The inner side of the same bone showing bevelling of edges.

shaped, resembling contact entrance wounds. The edges of the exit wound may be puckered or torn and everted, and broken pieces of contused, haemorrhagic subcutaneous fat may protrude through the defect. The edges are free from signs of burning, blackening or tattooing and there is no contusion or abrasion collar. When the weapon has been fired in contact with the bone, or at very close range, the exit wound is usually smaller than the wound of entrance. With increased range, the exit wound is larger than entrance wound. In a fatty person, the edges of both the wounds of entrance and exit may be everted due to protrusion of fat. They may also be everted in decomposed bodies (Table 8-8).

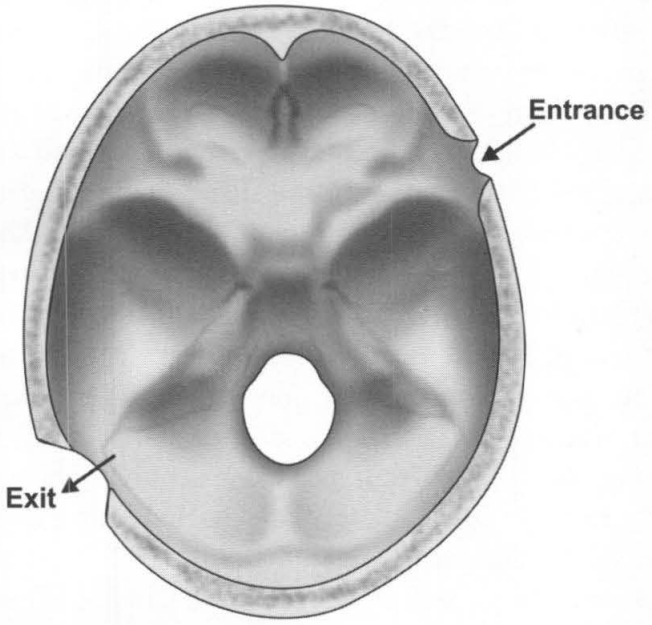


Fig. (8-30). Shooting appearances of entrance and exit wounds in skull.

Shored exit wounds: **If the skin at the exit wound is supported, the exit wound appears as a circular defect surrounded by a margin of abrasion resembling a wound of entrance (shored or supported exit wound)**. Many shored exit wounds are caused if a firm object, e.g., a belt, the waist band of trousers, etc., brassiere, collar and tie, is pressed against the body at the site of exit wound, or if the body is leaning against a hard surface, such as a wall, back of a chair or the floor if the person was lying down. In such cases, the skin crushed by the exiting bullet, produces an irregular, lopsided and large abrasion around or adjacent to the wound ("shored" exit wound) (Plate 11-61).

Table (8-8). Difference between wounds of entrance and exit

	Trait	Entrance wound	Exit wound
(1)	Size:	Smaller than the diameter of the bullet. In close discharge, skin is torn	Bigger than the bullet.
(2)	Edges:	Inverted.	Everted, puckered or torn.
(3)	Bleeding:	Less.	More.
(4)	Burning, blackening, tattooing:	May be seen around the wound.	Absent.
(5)	Contusion, abrasion and grease collar:	Present.	Absent.
(6)	Fat:	No protrusion.	May protrude.
(7)	Tissues within and around the wound:	May be cherry-red due to CO of explosive gases.	Absent.
(8)	Fibres of clothing:	Turned in and may be carried into the wound.	Turned out.
(9)	Lead ring or metal ring:	May be seen around the wound by radiological examination.	Absent.
(10)	Spectrography:	More metal is found around entrance wound, if bullet has only passed through soft tissues.	The exit wound may contain more metal, if a bone is struck nearer to it.

Variations in exit wounds: The variation in the shape and large size of the exit wound are due to: (1) The bullet tumbles in the body and fails to exit nose-end first. (2) The bullet is deformed. (3) The bullet breaks up in the tissues and exits as several pieces. (4) Fragments of the bone may be blown out of the body with the bullet. (5) The unsupported skin at the exit tends to tear and break into pieces.

Revolvers and automatic pistols cause similar wounds, but penetrating power of bullets of pistols is much greater, because of the greater velocity, and because of their being coated with hard metal.

X-ray Examination of Gunshot Wound Victims: It helps to: (1) locate the bullet or pellets, (2) locate bullet fragments or jackets **(Plate 11-62)**, (3) determine the break up pattern of the bullet, (4) show the track of the bullet **(Plate 11-63)**, (5) determine defects in bone, (6) locate air embolism accompanying large vessel damage by the bullet.

PECULIAR EFFECTS OF FIREARMS

(1) ATYPICAL ENTRANCE WOUND: (a) For a few microseconds after the bullet leaves the muzzle, there may be a "tail wobble" or "tail wag". This is partly responsible for the great tissue damage and the large atypical entrance wound at short range. (b) The gyroscopic effect of the bullet diminishes as it reaches the end of its flight, until it begins to wobble, and then to tumble, and produces large atypical wounds of entrance. A bullet travelling in an irregular fashion instead of travelling nose-on is called a YAWNING BULLET, (Fig. 8-31) and a bullet that rotates end-on-end during its motion is called a TUMBLING BULLET (Fig. 8-32).

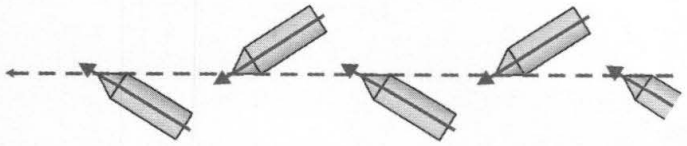


Fig. (8-31). Schematic depiction of "yaw" associated with missiles of very high velocities.

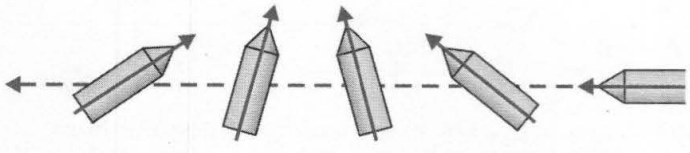


Fig. (8-32). Schematic depiction of "tumble" associated with missiles of very high velocities.

(c) RICOCHET BULLET: A ricochet bullet is one which before striking the object aimed at, strikes some intervening object first, and then after rebounding from these hits the object. The deflection of bullet is partly due to the obliquity with which it strikes and partly due to rotating motion on its axis. The bullet may ricochet before or after striking the body, and may produce a non-penetrating or a penetrating injury. Ricocheting of a bullet may occur with inferior firearms and low velocity bullets. **The bullet may be deformed and flattened before striking the skin.** This produces a large, irregularly-oval, triangular or cruciate entrance wound with ragged margins. **As the bullet loses gyrating movements, abrasion collar is absent. Burning, blackening and tattooing are also not seen.** Sometimes, a bullet may strike an object and tumble, and hit the body side on, producing an elongated wound of entrance like a keyhole. The path of a ricochet is completely unexpected. Particles of the substance against which the bullet is deflected or has struck, e.g., soil, fibres, paint, etc., may be found adhering to the bullet. The nose of the bullet may be found facing the entrance wound due to deflection in the body. When the velocity is lost, the bullet only produces an abrasion or contusion.

Internal ricochet: Sometimes, after passing through the brain, there is not enough energy left in the bullet to penetrate the skull. It may rebound from the inner table of the skull, like a billiard ball producing a second track. If it rebounds for a second time, a third wound track is produced **(Fig. 8-33)**.

(2) SINGLE ENTRANCE AND MULTIPLE EXITS: If the bullet splits up within the body and divides itself into three or four pieces, there will be only one entry hole, but several exits. The bullet striking a bone may break it and the fragments act as secondary missiles producing multiple exits. Sometimes, the jacket of a bullet may separate from the inner part upon impact on bone and each produce separate tracks.

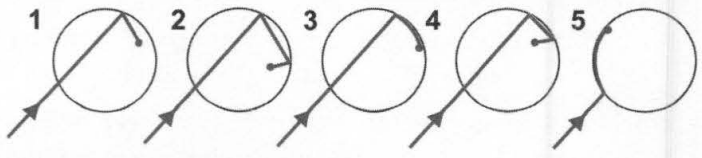


Fig. (8-33). Internal ricochet. (1) Single ricochet. (2) Double ricochet. (3) Inner tangential at contralateral side. (4) Inner tangential at contralateral side and ricochet. (5) Inner tangential at entrance side.

(3) MULTIPLE WOUNDS OF ENTRANCE AND EXIT FROM A SINGLE SHOT: A bullet may pass through an arm and the chest, so that four wounds result. A bullet passing through the chest or abdomen and thigh and lower leg produces six wounds. This occurs when the person is running or sitting in an unusual position. In such case, examination of clothing is very important.

(4) ENTRANCE WOUND IS PRESENT BUT BULLET IS NOT FOUND IN THE BODY: This occurs when the bullet (a) entering the mouth may be spit out, (b) entering the stomach may be vomited, (c) entering the gastrointestinal tract may be passed out in the faeces, (d) entering the windpipe may be coughed up.

(5) Unexplained Bullets in the Body: Occasionally, more bullets are found than there are entrance wounds. **This occurs due to defect in the weapon, or due to faulty ammunition, or with loaded firearm unused for several years. When such a weapon is fired, the bullet may fail to come out from the muzzle. When it is fired again, the second bullet may go off carrying the lodged bullet with it, and both the bullets may enter the body through the same entrance wound.** This is called a **tandem bullet or piggyback bullet** (tandem = one behind the other) **(Fig. 8-34)**. The bullets may separate within the body or before they hit the target. The features caused by flame, smoke and gunpowder may be diminished or absent and the wound may appear as if caused by long range fire. This is because the pushing force of the second bullet is directed backwards due to obstruction caused by first bullet impacted in the barrel.

Souvenir Bullet: If a bullet is present for a long time in the body, there is no fresh bleeding in the surrounding area. **A dense fibrous tissue capsule usually surrounds it.** A tiny scar indicates the original entrance wound.

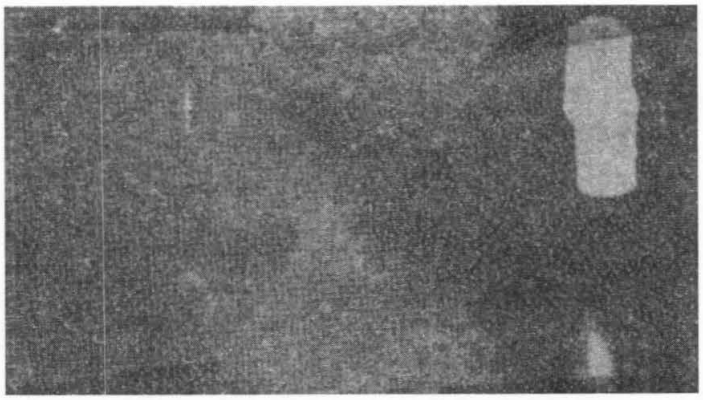


Fig. (8-34). Tandem bullets in the chest.

(6) FATALITIES WITH BLANK CARTRIDGES: A blank cartridge is one containing primer, gunpowder and wadding, but without a bullet or pellets. Wadding or gunpowder may cause laceration and may produce death from shock by pressure on nerves or by damaging large vessel, when blank cartridge is discharged close to the body. Distant shots with blank cartridge are harmless.

MEDICOLEGAL QUESTIONS

(1) Is the injury caused by discharge of firearm?

Firearm wounds are recognised by the appearance of clothing and body entrance and exit wound, the track of the bullet, and the presence of bullet or pellets and residual matter in the clothing, or around entrance wound and in the tissues.

(2) What kind of weapon fired the shot?

The kind of firearm can be determined by the size, shape and composition of the bullet, and examination of cartridge, shots and wad left in the body or found at the scene of the crime, and the appearances of wounds. With the shotgun, the appearances of the wound are characteristic. In muzzle-loading gun, the wad consists of plug of paper or cloth; in breech-loading gun of circular discs of felt or cardboard, from which the bore of the gun can be determined. Stains on the clothes or skin may show whether black or smokeless powder was used.

The firearms leave their signature on the cartridge case and on the bullet. **With all rifled firearms, the bullet is slightly larger than the barrel, and as it passes through the barrel, its sides are marked by the rifling of the barrel (primary markings).** They are most useful in identifying the make and model of gun involved. **The surface of the bullet is also grooved by irregularities on the inner surface of the barrel itself (secondary markings) which are specific for that particular weapon.** These irregularities (bar codes) are produced by the sticking of the particles of the bullet to the bore when shots are fired, and is known as **'metallic fouling'. Individual characteristics also result accidentally during manufacturing process, are usually microscopic in nature, and have random distribution. They are useful to identify one specific gun to the exclusion of all others (bullet fingerprinting).** The bullet found

in the body known as **crime bullet or exhibit bullet** is compared under a comparison microscope, with one fired from the suspected weapon known as **test bullet.** The suspected weapon is fired using the same brand and type of ammunition into a roll of wool, a bag of rags, a sand bag, box filled with cotton waste (bullet traps), water tank (bullet recovery tank), or against white blotting paper. Fresh pork skins, cleanly shaven are ideal for comparison with patterns on human skin. From the fired cartridge case, the caliber and type of cartridge and its manufacturer can be determined from the shape and size of the case and the stampings on the case head. Fired cartridge cases also may be identified with the firearm from which they are discharged by a study of the marks left on them by the weapon.

(3) FROM WHAT DISTANCE AND DIRECTION WAS THE SHOT FIRED?

The range of fire is determined by the presence or absence of the marks of smoke, flame, tattooing, etc., on or in the body of the victim. When the range is greater, it can be determined only approximately and with difficulty, from the nature of wounds and penetration. Test fire with suspect weapon using the same ammunition is useful for estimating the range. The direction of fire may be determined from the position of entrance and exit wounds and the track.

(4) WHEN WAS THE FIREARM DISCHARGED?

Tissue reaction to firearm injury is similar as for other types of injury. If black powder has been used, hydrogen sulphide may persist in the barrel for a few hours if breech is closed. The washings from barrels having discharged gunpowder are alkaline, contain nitrite, sulphate and thiosulfate. Smokeless powders leave a dark-grey deposit in the barrel of a recently discharged firearm. The mixture of gases of explosion has a peculiar smell, which can be noticed for several hours after the discharge of a gun.

DERMAL NITRATE TEST: It detects gunpowder residue (nitrates and nitrites). Melted paraffin is brushed on the surface of the hand. The wax is removed and the inner surface of wax cast is treated with diphenylamine or diphenylbenzedine reagent. A blue colour develops where residue is present. It is absolete, because a positive test is given if the hand is contaminated with nitrogenous compounds, e.g. urine, cigarette smoke, fertilisers, leguminous plants, etc.

(B) Neutron Activation Analysis.

(5) IS IT A CASE OF HOMICIDE, SUICIDE OR ACCIDENT? (A) THE POSITION AND DIRECTION OF THE WOUND: These are very important. For suicide, the sites of election are: (1) temple (about 60%), (2) center of forehead, (3) roof of mouth, (4) midline behind the chin, and (5) left side or front of chest.

SUICIDE: A suicide using a revolver or pistol, usually shoots himself in the right temple, the bullet passing almost horizontally or upwards and backwards through the head, and making its exit in the left parietal region. Sometimes, the gun is put into the mouth, nose, ear, or on the face or undersurface of the chin, and fired upwards into the brain. On the chest, the gun is fired into the cardiac region in a backward direction with a slight deviation upwards or downwards, either to the right or left. On the abdomen, the gun is fired into the epigastric region.

In rifle and shotgun, the butt is supported either by ground, and the weapon is fired by hand into the head, or the butt is supported against a wall and fired into the chest or abdomen. The person may attach some string or cord to the trigger and tie the loose end to his foot, lie down and discharge the weapon through the roof of his mouth. He may tie the cord to some firm structure and pull the barrel towards him, thus, firing the weapon or he may press the trigger with a stick or some similar object. The presence of blood stains on the hand which held the gun, or the presence of an impression of the trigger guard on one or more fingers indicates suicide.

HOMICIDE: In homicide, a great variety of wounds can occur depending upon the circumstances. If there is a scuffle, some of the wounds may be from close range, and the bullets may strike the body in various places and at different angles. If the victim runs, most of the entrance wounds will be on the back. If the victim rushes at the assailant, the entrance wounds are in front of the body. If the assailant is in a panic or under strong emotion, a number of shots may go wild or graze the skin. Wounds about the sides and limbs are suggestive of murder, if accident is excluded, and wounds about the back, and back of head are strongly suggestive of murder. In gang feuds, the victim may be surprised and shot by enemies at close range into the back of the head and neck, with the bullet tracks parallel or only slightly deviating.

(B) THE NATURE OF THE ENTRANCE WOUND: This is very important. A contact wound is possibly suicidal, unless found on a part of the body which is not easily reached by the deceased, e.g., back of the chest or neck. Suicides usually pull the clothes aside to expose the skin before shooting themselves. Bullets vertically entering fatal parts like heart or head, indicate suicide. Several methods (combined suicides), or several attempts of committing suicide e.g., hanging and shooting indicate suicide. A close or distant shot is rarely suicidal.

(C) THE NUMBER OF WOUNDS: Multiple wounds are usually homicidal, especially if they occur on the back

or sides of the body, or involve different regions of the body, or are non-contact shots. Occasionally, multiple suicidal wounds are seen involving a single region like the temple, chest or abdomen or a combination of two different regions. When multiple suicidal wounds are fired serially, the injuries by the first shot may not incapacitate the victim immediately, and the person can fire other bullets into his body.

(D) THE POSITION OF THE WEAPON: In suicide and accident, the weapon is usually found at the scene of the crime; in cases of murder, the weapon is not found near the victim. If the weapon is strongly grasped in the hand of the victim due to cadaveric spasm, it is a strong presumptive evidence of suicide. In some cases of homicide, the scene may be altered to simulate suicide in order to conceal the crime. The assailant after producing a contact wound on the anterior surface of the body or an accessible part of head, may place the weapon in the hand of the deceased. In such cases, the thumb is usually found under and not around the gun handle, and the weapon is not tightly grasped.

(E) MOTIVE: When a person commits suicide, he usually has a motive, and he may leave a note or make preparations to kill himself.

(F) THE SCENE: Fingerprints on the weapon, doors, etc., are useful. If the gun is present at the scene, its position in relation to the body, its make, model, calibre and description of the ammunition should be noted. If they are embedded in the walls, ceiling or furniture, they should be extracted and retained. The place in which the body is found, e.g., in a room in his own house, the state of the room whether showing signs of the struggle or not, the condition of the doors and windows whether locked or not should be noted. If the body is found in the open, a search for footprints and marks of struggle must be made.

(G) SEX: It is very rare for females to commit suicide by shooting, and rare for persons who are not used to firearms.

ACCIDENTAL WOUNDS: They are comparatively rare and usually single. Most of the accidents are caused by carelessness or misuse. Accidents occur when loaded weapons are handled carelessly or carried without safety catch, or when the victim slips while examining, loading or cleaning the weapon. The wounds are found on the front of the body and frequently directed upwards. Sometimes, the victim is shot unintentionally by a person who is ignorant of firearms or careless in their use. Occasionally, when the police shoot on law breakers, an innocent bystander is struck by bullets. Hunting accidents also occur.

BOMB EXPLOSION WOUNDS

A bomb is a container filled with an explosive mixture and missiles, which is fired either by a detonator or a fuse. When an explosion occurs, the explosive material produces a large volume of gas and releases very large amount of energy. Pressures of up to thousand tons per sq. cm. can be produced. A person can be injured by an explosion in a number of ways.

(1) Disruptive Effects: If the victim is almost in contact with a large bomb, he may be blown to pieces, e.g., when the victim is carrying it. The pieces can be scattered over an area of 200 metres radius. Many parts of the body are never found. A bomb exploding on the ground may cause severe damage or traumatic amputation of the legs. When the victim is a few metres away or with smaller explosions, disruption is usually limited to mutilation of a localised area.

(2) Burns: The temperature of the explosive gases can exceed 2000°C., and the radiated heat can cause flash burns. It burns nearby objects and clothing. The flame causes extensive burns, which involve irregular areas of skin to a different degree. Tight clothing protects, so that beneath collars, bras, waistbands, socks and shoes the skin may be quite normal.

(3) Air Blast: An explosion produces a 'shock wave' which spreads concentrically from the site of explosion at about the speed of sound (1120 feet/sec). The shock wave can throw the victim against a wall, or toss him through the air causing blunt force injuries. The clothes may be blown off by the blast. The shock wave passes through the body. The homogeneous tissues like liver and muscle are not damaged. The tympanic membrane ruptures most commonly with haemorrhage in the ear. Damage to cochlea is more frequent. **Lungs show subpleural patchy haemorrhages, often in the line of ribs, rupture of alveolar septa and bullae at the margins.** Desquamated alveolar and bronchial epithelium is seen lying free. This causes reactive pulmonary edema and blood-stained froth is found in the air-passages, and later bronchopneumonia. This specific pulmonary injury of air blast is called **'blast lung'.** Intracranial haemorrhage, contusions of the brain, aortic and heart injuries, pneumothorax, subperitoneal haemorrhage,

ruptured stomach and bowel may occur. Death may occur from systemic air embolism from air which enters the pulmonary veins after blast damage to the lungs. When the explosion is in the water, the pressure changes are called **underwater blast.** The physical changes are similar to those of explosion in air. Injuries occur mostly in gastrointestinal tract and less commonly in lungs. **Solid blast** refers to a wave of energy that spreads through a rigid structure when an explosive is detonated near it and people in contact with its structure can be injured. Tanks constructed of steel and warships conduct shock wave well and cause solid blast injury. The injuries are mostly skeletal.

(4) Flying Missiles: The blast may drive multiple fragments of bomb or pieces of nearby objects, e.g., **gravel, glass, wood, brick, plaster, etc. through the air into the skin and cause bruises, abrasions and puncture-lacerations intimately mixed on the skin. This triad of injury is diagnostic (Fig. 8-35 and Plate 11-66).**

Fig. (8-35). Bomb explosion injuries showing contusions, abrasions, puncture lacerations and tattooing.

(5) Falling Masonry: When a building is destroyed by a bomb blast, the persons inside sustain multiple injuries and die of traumatic asphyxia.

(6) Fumes: If a bomb explodes in a confined space, enough CO is produced to cause asphyxia.

Mechanical Explosion: Mechanical explosion occurs when a steam boiler bursts due to increased pressure. Heat and large volumes of gas are produced. The effects are similar to those of chemical explosion.

CHAPTER

9

Regional Injuries

HEAD INJURIES

SCALP: Bruising of the scalp is better detected by touch than by sight. Multiple contusions of the scalp may join together, and as such it is often difficult to determine the number of blows inflicted. Its firm edge often feels like the edge of a depressed fracture of the skull. A scalp wound by a blunt weapon frequently resembles an incised wound, and as such the edges and ends should be carefully examined with a magnifying lens.

BLACK EYE (PERIORBITAL BRUISING): It is caused by: (1) Direct blow in the front of orbits, bruising lids. (2) Injury to the forehead, the blood tracking down under the scalp. (3) Fracture base of the skull in the anterior fossa, the blood leaking through cracked orbital plates.

SKULL

Fractures: Fractures may be caused by direct or indirect violence. Direct injuries may be caused by: (1) Compression as by midwifery forceps, or crushing of the head under the wheel of a vehicle. (2) An object in motion striking the head, e.g., bullet, brick, masonry, machinery, dagger, etc. (3) Head in motion striking an object, as in falls and traffic injuries. Indirect injury of the skull occurs from a fall on the feet or buttocks.

Types of Fracture: (1) Fissured Fractures: These are linear fractures or cracks in the bone, involving the whole thickness of the bone, or inner or outer table only (Fig. 9-1). It may be present alone or associated with other types. **They are caused by forcible contact with a broad resisting surface like the ground, blows with an agent having a relatively broad striking surface or from a fall on the feet or buttocks.** When a blow is struck on the side and the head is free to move, the fracture starts at the point of impact and runs parallel to the direction of the force. If the head is supported when struck, the fracture may start at the counter-pressure, e.g., in bilateral compression, the fracture often starts at the vertex or more commonly at the base. In both cases, the line of the fracture runs parallel with the axis of compression. Linear fractures do not tend to cross bony buttresses, but may cross points of weakness. A fracture line tends to follow an irregular course and usually is of hair's breadth. Sometimes, a fracture line reaches a suture and follows its course for a while, and then starts again on its own way. The fracture line is often red, but usually there is no haemorrhage.

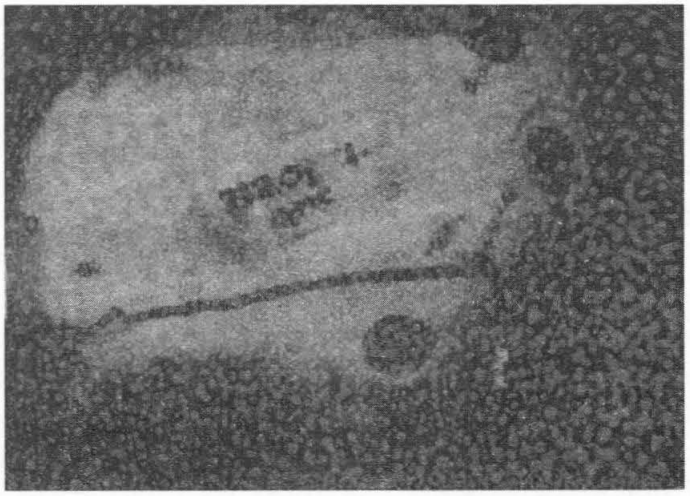


Fig. (9-1). Fissured fracture in the left parietal and occipital bones and two burr holes.

(2) Depressed Fractures: They are produced by local deformation of the skull. **Localised depressed fractures are caused by blows from heavy weapon with a small striking surface, e.g.,** stone, stick, axe, chopper, hammer, etc. The outer table is driven into the diploe, the inner table is fractured irregularly and to a greater extent and may be comminuted. In this, pieces of fractured bone are driven inwards into the skull cavity**. They are also called "fractures a la signature" (signature fractures), as their pattern often resembles the weapon or agent which caused it (Plate 12-68).** Sometimes, the depressed fracture may involve the outer table only. Rarely, only the inner table may be fractured under the site of impact, leaving intact the outer table. **A violent blow with the full face of a weapon completely detaches almost the same diameter of the bone, which is driven inwards.** A less violent blow, or an oblique blow, may produce a localised fracture with only partial

depression of the bone. When a hammer is used, the fracture is circular or an arc of a circle, having the same diameter as the striking surface. The part of the skull which is first struck shows maximum depression (Fig. 9-2).

Multiple blows on the head: The first blow to the skull will weaken the structure and subsequent blows may cause a degree of damage out of proportion to the force used, e.g., a second blow in an area of skull already fractured, may cause widespread collapse of the skull in that area, and the fragments of bone and the weapon itself may be driven far into the brain.

(3) Elevated Fractures: One end of fractured fragment is elevated over the surface of skull and the other end is depressed into cranial cavity. **It is caused by a blow from heavy sharp weapon which elevates the fragment by lateral pull of the weapon while retrieving it.**

(4) Comminuted Fracture: In a comminuted fracture, **there are two or more intersecting lines of fracture which divide the bone into three or more fragments (Fig. 9-2). They are caused by a fall from a height on a hard surface, vehicle accidents and from blows by weapons with a large striking surface, e.g.,** heavy iron bar, an axe, thick stick, etc. They may result from a kick by an animal or by a bullet. It is often a complication of fissured or depressed fractures. **When there is no displacement of the fragments, it resembles a spider's web or mosaic.** Fissured fractures may radiate for varying distances from the area of comminution (fragmentation). When the force is great, the broken pieces of bone are displaced and some may enter the brain and others may be lost.

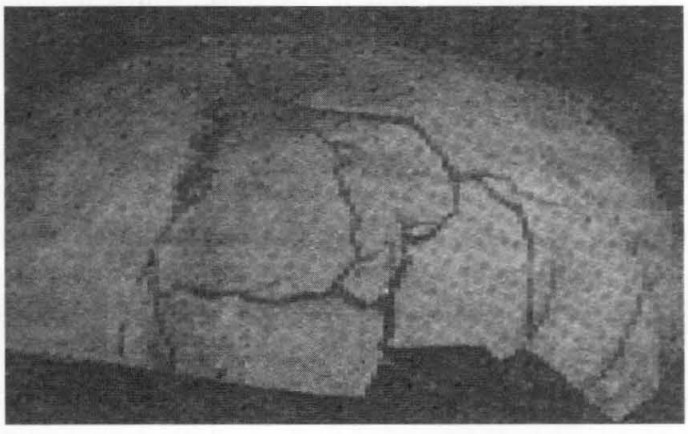


Fig. (9-2). Comminuted fracture of skull.

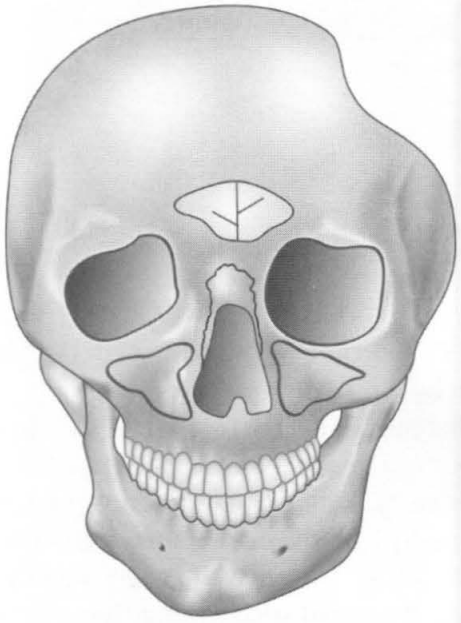


Fig. (9-3). Pond fracture.

(5) Pond or Indented Fracture: This is a simple dent of the skull, which results from an obstetric forceps blade, a blow from a blunt object, or forcible impact against some protruding object. **They occur only in skulls which are elastic, i.e., in the skull of infants.** The inner table is not fractured but fissured fractures may occur in the outer table around the periphery of the dent. In an infant, a blow often produces only a dent like that seen in a damaged Ping-Pong ball (Fig. 9-3).

(6) Gutter Fractures: They are formed when part of the thickness of the bone is removed so as to form a gutter, e.g., in oblique bullet wounds. They are usually accompanied by irregular, depressed fracture of the inner table of the skull. The dura mater and brain may be torn.

(7) Ring or Foramen Fracture: It is fissured fracture which encircles the skull in **such a manner that the anterior third is separated at its junction with the middle and posterior third. But, usually the term is applied to fracture, which runs at about 3 to 5 cm outside the foramen magnum at the back and sides of the skull, and passes forwards through the middle ears and roof of the nose, due to which the skull is separated from the spine (Fig. 9-4).** They are rare and occur after falls from a height on to the feet or buttocks. In very severe cases, the spine may be driven into the skull cavity, and the vault of the skull may burst open (explosive type), giving the impression of an original massive fracture. A severe

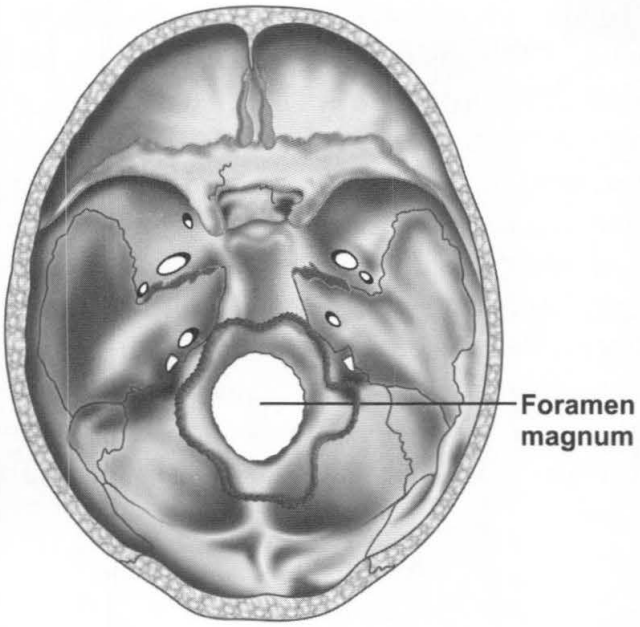


Fig. (9-4). Ring fracture.

blow to the vertex may also cause a ring fracture, but in this case the fracture of the vault will be depressed. A forceful blow on the chin in traffic accident may produce ring fracture.

(8) Perforating Fractures: These are caused by firearms and pointed sharp weapons like daggers or knives and axe. The weapon passes through both tables of the skull leaving a clear-cut opening, the size and shape of which corresponds to the cross-section of the weapon used.

(9) Diastatic or Sutural Fracture: Separation of the sutures occurs only in young persons due to a blow on head with blunt weapon. It may occur alone but often is associated with fracture.

(10) Expressed Fractures: These occur as **massive fragmentation/shattering of skull where some pieces may be found outside the head.** They occur due to massive trauma often involving contact or close range firearm injuries or due to bomb blasts.

Countrecoup fracture: Fracture of the skull **occurring opposite to the site of force is known as contrecoup fracture.** This usually occurs when the head is not supported. This is explained by the sudden disturbance in the fluid brain content which transmits the force received to the opposite side, where the thrust of violent motion impacts against the cranial wall, which is unable to absorb this degree of disturbance.

AGE OF SKULL INJURY: Healing occurs without the formation of visible callus. The edges of fissured fracture stick together within a week. The edges are slightly eroded and the inner surface of the skull may show pitting or deposition of lime salts in fourteen days. The edges become slightly smooth and bands of osseous tissue run across the fissure in three to five weeks. When the edges are not in apposition, they become quite smooth in three months, or partially healed if close together. If there is much loss of bone, the gap is filled only with fibrous tissue.

MECHANISM OF CEREBRAL INJURY

Brain injuries may be caused by: (1) Penetration by a foreign object, such as knife, bullet, etc., or fragments of skull in a depressed fracture. (2) By distortion of the skull. When a localised segment of the skull undergoes deformation, shear strains may develop in the brain tissue underlying the indentation, and a contusion may be produced in the surface layers of the brain tissue. If a fracture occurs, pieces of bone may penetrate the dura and lacerate the brain. (3) Due to movement of the brain in relation to the skull.

Contusions of the Brain: When a localised segment of the skull undergoes deformation at the moment of impact, shear strains may develop in the brain tissue underlying the indentation, and a zone of contusion may be produced in the surface layers of the brain **(Fig. 9-5)**. They may occur without injury of the skull, but often there is a fracture of the skull. Most often they occur in cerebral and cerebellar cortices and subcortical areas. Deeper structures, e.g., basal ganglia, midbrain and brainstem are contused from impacts in forehead and vertex **(Fig. 9-5 and Plate 12-69 to 12-72)**.

Types: Contusions found in deeper structures of the brain along the line of impact are called **intermediary coup contusions.** Contusions caused by fracture of skull are called **fracture contusions.** Contusions in the cortex and white matter of frontal and central convolutions are called **gliding contusions.** Contusions in the cerebellar tonsils and the medulla oblongata are called **herniation contusions**.

Cerebral lacerations: They are traumatic lesions in which there is loss of continuity (tearing) of the substance of brain. When the parenchyma is completely disorganised, it is termed pulpefaction. Lacerations are usually seen underneath skull fractures. All penetrating injuries produce lacerations of brain. Lacerations may also be produced without

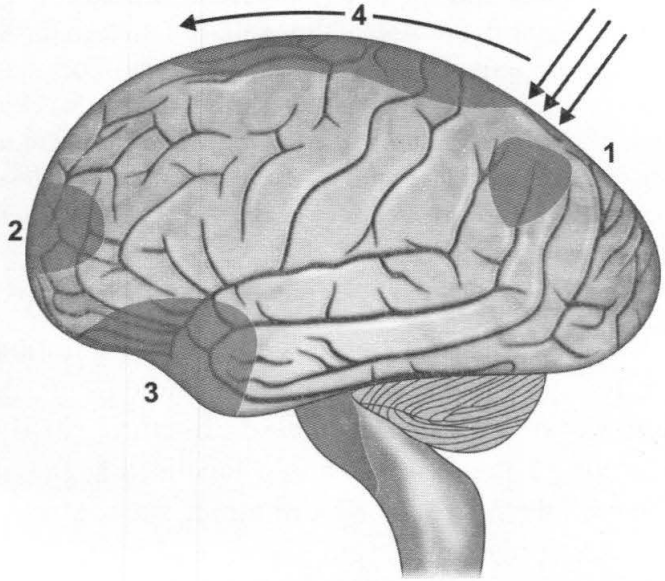


Fig. (9-5). Contusion and laceration of the brain. (1) Coup injury. (2) Contrecoup injury.(3) Dashboard injury in the region of the sphenoidal ridge. (4) Avulsion injury along the superior border of the hemisphere (the rotary vein).

fracture of skull, when they are usually found in regions where the brain is in contact with projecting buttresses and ridges on the inner surface of the skull, e.g., the temporal poles and orbital surface of the frontal lobes.

CONTRECOUP LESIONS: Coup (blow. impact) means that the injury is located beneath the area of impact and results directly by the impacting force (Fig. 9-6). Contrecoup means that the lesion is present in an area opposite the side of impact (Fig. 9-7). A certain amount of shear may occur below the point of impact, particularly if the skull is fractured, which accounts for the coup. A much greater shear strain develops as a result of the rotation of the skull, and because the changes in the rotational velocity are usually greater at the pole opposite to the point of impact, contrecoup injuries are more extensive. If a person is lying on the ground or against some unyielding surface, a heavy blow on the upper temporal or parietal area may cause contrecoup injuries in the contralateral temporal or parietal cortex. Contrecoup injuries are rare before the age of three years. In some cases, there may be no coup damage at all, only contrecoup.

Mechanism: Contrecoup injury is caused when the moving head is suddenly decelerated by hitting a firm surface, e.g., striking the head on the ground during a fall, usually seen in traffic accidents (deceleration injury). The sudden arrest of the head results in the brain which is still in motion, striking the arrested skull. A blow to the head causes the skull to move forward, but the brain lags behind for a brief period and the skull strikes the brain (acceleration injury). Another factor responsible for contrecoup injury is formation of a cavity or vacuum in the cranial cavity on the opposite side of impact as the brain lags behind the moving skull. The vacuum exerts a suction effect which damages the brain. **Occipital injuries**

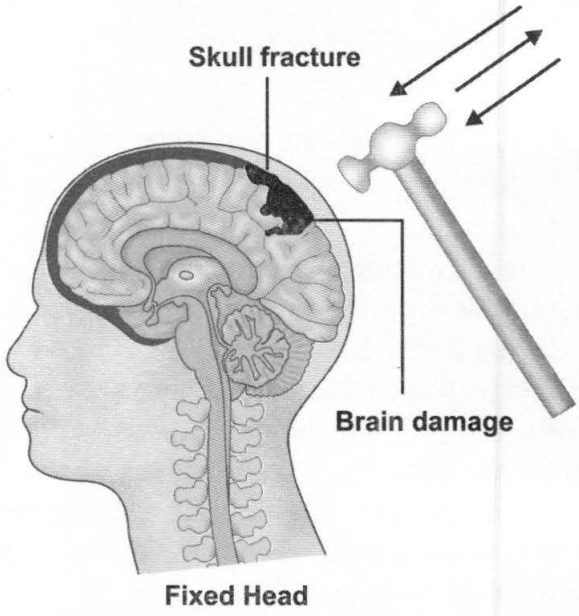


Fig. (9-6). Mechanism of coup injuries of the brain.

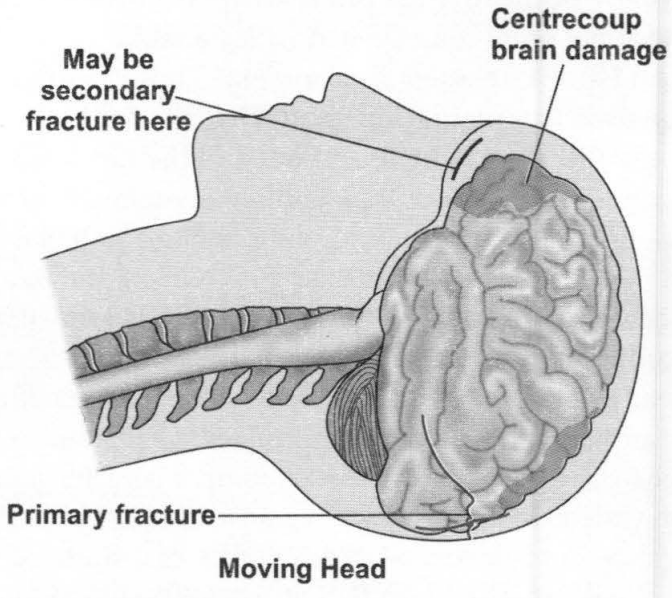


Fig. (9-7). Mechanism of contrecoup injuries of the brain.

produce severe and extensive contrecoup lesions in the frontal region. The irregular bony prominences, especially of the orbital and cribriform plates, and the lesser wings of the sphenoid, contuse or lacerate the base of the frontal lobes and the tips of the temporal lobes, sometimes with fracture of orbital plates. A blow at the front of the head may rarely damage the inner and lower parts of the back of brain by contact with the edges of the tentorium. A fall on to the side of the head may cause a fracture of that side, and contusion of the opposite side of the brain. **A blow to the head produces coup contusions, while contrecoup contusions are either small or absent. A fall on the head produces contrecoup contusions while coup contusions are small or absent (Figs 9-6 and 9-7). Contrecoup injury is seen in skull, brain, liver, heart and lungs.**

CONCUSSION OF THE BRAIN

Concussion is a state of temporary unconsciousness (due to partial or complete paralysis of cerebral function), **due to head injury, comes on immediately after injury, is always followed by amnesia, and tends to spontaneous recovery.** True concussion may last for seconds or minutes.

Mechanism: Cerebral concussion occurs due to acceleration/deceleration of the head. **Cerebral concussion may be produced by direct violence to the head, or by indirect violence as a result of violent fall upon the feet or buttocks, by an unexpected fall on the ground in traffic or industrial accidents.** The violent head movement causes shearing or stretching of the nerve fibres and axonal damage. **Severe injuries occur in coronal head motion only.**

Features: There are many theories as to the cause of concussion: vasomotor disturbances, impaction of the brain into the foramen magnum or tentorial opening, but the most **acceptable hypothesis is "diffuse neuronal injury"**, a functional abnormality of the nerve cells and of their connections. Experimental evidence suggests that in concussion, direct damage occurs to the brainstem reticular formation. As consciousness gradually returns, there is a period during which the person appears to be lucid and in touch with his surroundings, but in fact he is not. **He behaves automatically and not rationally or responsibly. He may commit some violent or criminal act. The patient has no recollection of accident or injury, although he can usually recall events up to or within a few minutes of the occurrence.** A post-traumatic amnesia may range from minutes to days, and its duration is usually proportional to the severity of the injury. Death may occur without the patient regaining consciousness, or he may recover partially and then die suddenly, from concussion of vital cerebral centres. In cases of recovery, **post-concussion syndrome** may follow with headache, giddiness, nausea, vomiting, insomnia and mental irritability. **Autopsy may not show any change,** but in some cases petechial haemorrhages may be found in the brain.

Head injuries and acute alcoholic intoxication: A person may be confused and disorientated after a head injury which may simulate acute alcoholic intoxication (**Table 9-1**).

Admission to hospital after head injury: A person who has sustained head injury, should be admitted in hospital for observation for at least 24 to 36 hours.

Table (9-1). Difference between drunkenness and concussion

	Trait	Drunk	Concussed
(1)	Face:	Suffused; flushed, warm.	Pale, clammy.
(2)	Pupils:	Contracted in coma, dilate on external stimuli and contract again. Reaction to light sluggish.	Contracted or unequal.
(3)	Pulse:	Fast, bounding.	Slow, feeble.
(4)	Breathing:	Sighs, puffs, eructates.	Shallow, irregular, slow.
(5)	Memory:	Confused.	Retrograde amnesia unrelieved by time.
(6)	Behaviour:	Uncooperative, abusive, unresponsive, arrogant, talkative.	Cooperative, quiet.

CEREBRAL OEDEMA: Cerebral oedema occurs due to a localised or diffuse abnormal accumulation of water and sodium, which increases the volume of the brain. It is caused due to increase in the intravascular pressure, increased permeability of the cerebral vessels, and decrease in plasma colloid osmotic pressure. Focal oedema is almost invariably associated with and secondary to contusions and lacerations of the brain. Focal oedema in the brainstem is usually fatal. Generalised cerebral oedema occurs with diffuse brain injury (Plate 15-85).

INTRACRANIAL HAEMORRHAGE

(1) EXTRADURAL HAEMORRHAGE

Causes: It is **caused almost exclusively due to trauma**. At the moment of impact, the skull moves relative to the dura underneath it, and the dura is stripped from the bone. This produces an empty extradural space at the site of trauma. A blood vessel may be injured at the same time. The vessel injured depends upon the site of trauma. (1) **A blow over the lateral convexity of the head may injure the middle meningeal artery.** Less commonly, the posterior meningeal artery near the foramen magnum or anterior meningeal artery near cribriform plate are injured. (2) A blow over forehead involves the anterior ethmoidal artery. (3) A blow over the occiput, or low behind the ear, may tear the transverse sigmoid sinus and produce posterior fossa haematoma. (4) A blow on the vertex may cause haemorrhage from sagittal sinus. (5) Venous extradural haemorrhage accompanies fracture of skull (usually occipital) and is due to bleeding from the diploic veins.

Occurrence: It is the **least common type of meningeal bleeding, and is seen in one to three per cent of cases of head injury**. These haemorrhages are not common in the first two years of life, due to the greater adherence of the dura to the skull and the absence of a bony canal for the artery, but are **common in adults between 20 and 40 years.** Haemorrhages may occur due to fall from a small height, or on being struck by a moving object, or after a minor accident. Bleeding may continue for many hours or even a day after the injury. In 90% cases the fracture is of fissured type, but sometimes is depressed. Rarely, the haemorrhage is found without any fracture of the skull or any external injury to the head.

Blood Clot: In almost all cases, the haematoma is directly under the site of surface injury. The clot is localised, presses the dura inwards and causes a localised concavity of the external surface of the brain. The clot has typical limitation due to the dural attachments at the suture lines. **The clot is oval or circular, rubbery consistency, reddish-purple, about 10 to 20 cm in diameter, 2 to 6 cm thick, weighs 30 to 300 g, and is adherent to the dura mater.** The clot is usually in the tempero-parietal area or in the frontotemporal or parieto-occipital region **(Plate 13-73)**. The haematoma cannot be contrecoup unless the skull has been grossly deformed. If it is bilateral, then trauma has been bilateral, or a middle structure, such as the sagittal sinus has been injured. Usually, **hundred ml. is the minimum to cause death.**

Clinical Features: In a typical case, there is a history of head injury, which causes the bleeding and will usually cause temporary unconsciousness. In about 25% of cases, there is no initial unconsciousness. This is followed by a period of normal consciousness, the **"lucid interval" of few hours to a week. Lucid interval is seen only in 30 to 40% cases.** Lucid interval is not seen if the injury to the brain is sufficiently great, because of the overlapping of unconsciousness due to the brain injury and due to the pressure of epidural haemorrhage. **As the pressure on the brain increases, the patient first becomes confused and may appear to be drunk.** With increasing pressure, sleep and coma occur. The usual cause of death is respiratory failure due to compression of the brainstem. Tentorial herniation occurs. **Chronic extradural haematomas are rare**.

(2) SUBDURAL HAEMORRHAGE: This occurs in the subdural space.

Causes: (1) Rupture of bridging or communicating veins. (2) Rupture of inferior cerebral veins entering the sinuses at the base of the skull. (3) Rupture of dural venous sinuses. (4) Injury to cortical veins. (5) Lacerations or contusions of the brain and dura. (6) Re-injury of old adhesion between the brain and the dura. (7) Secondary to disease, e.g., cerebral neoplasm, cerebral aneurysms or blood disorders. (8) Drugs such as dicoumarol, warfarin and heparin can produce subdural haematoma, usually with but sometimes without a history of trauma.

Occurrence: Subdural haemorrhage may occur from relatively slight trauma, often insufficient to

cause unconsciousness and usually not producing fractures of the skull, and may be associated with contrecoup contusions. **They may occur after fights or falls, and are especially likely to be found in alcoholics, old persons and in children.**

Features: It is frequently seen over the upper lateral surface of the cerebral hemispheres. It is most commonly supratentorial **(Plate 13-74 and 13-75). They usually appear as thick layers of blood over the superior surface of the brain, with thin layers over the base of the brain in the anterior and middle cranial fossae. It is essentially venous or capillary and not arterial**. The volume of the blood varies from a few drops, or a thin-layered effusion to 150 ml. or more.

Types: It is divided into three types according to the time of onset of symptoms after the injury. (1) In the **acute type**, haemorrhage occurs from smaller bridging veins, immediately and very rapidly after the trauma. When there is a **lucid interval**, this may be **longer than the average 4 hours of extradural haemorrhage**. (2) In the **subacute type**, bleeding occurs from smaller bridging veins, the symptoms develop from several days to two to three weeks after injury, due to the pressure of the haematoma. The clot contains some dark fluid with formation of a thin peripheral membrane. (3) **Chronic type** (pachymeningitis haemorrhagica interna chronica) results from slight trauma in which symptoms appear some weeks or months later.

(3) SUBARACHNOID HEMORRHAGE:

Causes: (1) Rupture of a saccular Berry aneurysm. Blood accumulates rapidly on the undersurface of the brain **(Plate 13-77). It may occur due to sudden rise of blood pressure due to emotional stress, such as sudden exercise, sexual intercourse, etc. Alcohol also results in aggressive behaviour and a fight, resulting in ruptured aneurysm.** Berry aneurysms are rare in children, and increase in frequency with age. (2) Rupture of vessels on the surface of cerebral hemispheres. (3) Angiomas and arteriovenous malformations. (4) Lacerations and contusions of the brain and the pia-arachnoid. (5) Tears of the ventricular ependyma. (6) Injuries to the side of the upper neck and jaw region, producing damage to the vertebral artery, which causes basal subarachnoid haemorrhage. (7) Asphyxia. (8) Blood dyscrasias, leukaemias, etc. **Minimum trauma may precipitate haemorrhage**.

Alcoholism: In acute alcoholism, traumatic subarachnoid haemorrhage is more common due to loss of muscular coordination, resulting in excessive rotational forces within the head, and also possibly due to increased bleeding from congested vessels, which do not contract down, and the bounding pulse of the drunken man.

Features: This is the most common form of traumatic intracranial haemorrhage. In all cases of significant brain injury, some degree of subarachnoid haemorrhage is found. It may be the only evidence of head injury, but is often seen in association with other intracranial haemorrhages, with brain injuries, and with skull fractures. Thin-layered subarachnoid haemorrhage, usually along the sulci is common in head injuries. **It is mostly venous. In the mild form, the subarachnoid haemorrhage is present as splashes of haemorrhage over the areas of contusion. In most cases, it is diffuse overlying the cerebral hemispheres (Plates 13-76, 13-78 and 14-79).** Mild or moderate subarachnoid haemorrhage does not produce any significant damage. **A slightly yellow discolouration of the leptomeninges is seen as the subarachnoid haemorrhage becomes older.** It can be unilateral or bilateral, localised or diffuse. It is usually found over the orbital surface of the frontal lobes and the anterior third of the temporal lobes. In most severe head injuries it is present over the greater part of both hemispheres and is often accompanied by subdural bleeding. It spreads into the basal cisterns. The **blood mixes with the C.S.F.** and may be distributed over the whole of the brain.

(4) INTRACEREBRAL HAEMORRHAGE:

Causes: (1) Capillary haemorrhages are found in softening due to anoxia or arterial thrombosis or sinus thrombosis, in blood dyscrasias, in fat embolism and in asphyxial states. (2) Angioma or malignant tumour of the brain. (3) Hypertensive cerebral vascular disease. (4) Laceration of the brain. (5) **Spontaneous haemorrhages in the region of the basal ganglia by rupture of a lenticulostriate artery is common in middle-aged and elderly persons.** (6) Blow on the head, with or without fracture of the skull.

Most traumatic haemorrhages occur at the time of the accident, but bleeding is often slow and in the nature of oozing from venules or capillaries. **Haemorrhage into the brain due to trauma usually**

occurs near the surface. Isolated haemorrhages in the frontal or occipital lobes are more likely to be due to trauma (Plate 14-80 to 14-83). A single deep-seated haemorrhage is usually due to some disease.

Violence or Disease: Intracranial haemorrhage may be due to violence, disease or the effects of injury upon disease. When disease is present, sudden rise of blood pressure, due to physical exercise or excitement, e.g., alcohol, scuffle, assault, etc., may rupture vessels and precipitate haemorrhage. The commonly found diseases are cerebral aneurysm, cerebral tumours, especially angiomata, degeneration of cerebral arteries, syphilis, and evidence of hypertension. **Intracranial haemorrhage due to violence may occur without any fracture of the skull or wound of the scalp. Extradural haemorrhage is always caused by mechanical violence. Subdural haemorrhage is almost always traumatic in origin, but may be caused by local inflammation. Subarachnoid hemorrhage often occurs spontaneously from rupture of congenital aneurysms of blood vessels in the circle of Willis.**

Amnesia following Head Injuries: Amnesia following head injuries is quite common and is usually associated with concussion. The memory of distant events tends to return before the memory of more recent events. Permanent retrograde amnesia may vary from a period of seconds up to seven days. In cases recovering from concussion, events which occurred just before the injury are sometimes remembered indistinctly during the period of confusion, but there will be complete amnesia for these events after the return of complete consciousness. As such, the patient may make false accusations. Retrograde amnesia may also occur in injuries in which there is no loss of consciousness.

Post-traumatic Automatism: Automatism refers to the behaviour that occurs when a person is unconscious and unaware that the act is taking place. It is intimately associated with amnesia. After an accident, the patient may speak and act in purposive manner, but does not remember them afterwards.

Anterograde amnesia is characteristically seen in post-traumatic head injury.

Cause of death in head injury: (1) Raised intracranial pressure due to haematoma, contusions, lacerations, infarction or swelling is the common cause. (2) Disseminated intravascular coagulation. (3) Ischaemic brain damage. (4) Fat embolism.

VERTEBRAL COLUMN

FRACTURES OF THE SPINE: Causes: (1) Direct violence, and (2) indirect violence, as by forcible bending of the body or by a fall on buttocks or feet. Hyperflexion is the most common mechanism of fracture of spine. Falling from a height, diving and being thrown from automobile are the common causes. The common sites of fracture are upper and lower cervical regions and junction of thoracic and lumbar segments.

SPINAL CORD

Spinal cord can be injured by penetrating wounds, and by fracture and fracture-dislocation of vertebrae. The common sites of injury to the cord in order of frequency are: the lower cervical region, the thoraco-lumbar junction and the upper cervical region. Falls on the buttocks and the lifting of heavy weights may cause protrusion of an intervertebral disc and may compress adjacent spinal nerve root.

Concussion of the Spinal Cord: This commonly occurs in railway and motor car collisions, and is known as **'railway spine'**. It occurs from blows, compression from dislocation or fracture of the vertebrae, damage by effusion of blood, fall from a height or a bullet injury. It produces temporary paralysis, affecting the arms and hands or bladder, rectum, or lower extremities.

Death occurs immediately if the cord is damaged above the level of fourth cervical vertebra.

RIBS: Indirect violence through compression of the chest produces multiple and often bilateral fractures along the mid-axillary line and in the region of the posterior angle of the ribs, and the fragments usually bend outwards. The middle ribs from fourth to eighth are usually fractured. In fractures due to direct violence, the fragments are often driven inwards and lacerate the underlying structures. In a fall from a height, or when run over by a motor car or bullock cart, or compression of the chest by the knees or elbows, the ribs are fractured symmetrically on both sides, in front near the costal cartilages and at the back near the angles.

LUNGS: Compression of the chest or blows from a blunt weapon produce contusions or lacerations. If the impact is severe, there may be internal laceration of the tissue, either as multiple small areas of damage or there may be a large, irregular cavity 'traumatic cavitation'. Lacerations are usually produced by the penetration of the lung tissue by fractured rib ends.

HEART: Contusions and lacerations of the heart may be caused by direct violence on the chest or by compression of thorax, or when a driver is forcibly thrown against the steering wheel. This may or may not be associated with external injury or fracture of the ribs or sternum. Blunt cardiac trauma insufficient to produce gross or microscopic damage, can cause ventricular fibrillation or asystole (inhibition). Cardiac contusions are usually seen on the anterior surface of either ventricle or the interventricular septum. Contrecoup contusions of the heart are seen over the posterior wall of the left ventricle. They are seen in traffic accidents in which driver is thrown forward against the steering wheel and the heart is compressed against the vertebrae.

STOMACH AND INTESTINES: Compression injuries produce contusions or lacerations. Small intestine is more commonly injured by forces of compression than the stomach and the large intestine. Traction forces cause displacement of the stomach and intestines, but may stretch and rupture the attachment of the stomach or intestines, e.g., the mesentery may be torn near its intestinal attachment as a result of displacement of the small intestine. The stomach distended with food or diseased from ulcer or cancer is easily ruptured by blunt violence, usually at the pyloric end and the greater curvature.

LIVER: It is the most frequently damaged abdominal organ, and is second only to the brain in overall visceral susceptibility. The liver is commonly ruptured by a blow, kick, crushing motor accidents, fall or by a sudden contraction of the abdominal muscles. Signs of external injury may or may not be present. Contusions of liver may be difficult to recognise. Mild degree of external violence may rupture the liver if it is diseased, e.g., fatty metamorphosis, abscess formation, malaria, bilharziasis.

SPLEEN: Penetrating wounds of the spleen are less common than those of liver, but bleeding is more profuse. Compression forces produce lacerations. Traction forces may tear the spleen from its pedicle. The spleen is ruptured usually in its concave surface. Death from rupture of spleen is usually rapid, due to profuse haemorrhage. A relatively mild trauma or even the contraction of the abdominal muscles may predispose the spleen to rupture when it is diseased and enlarged, e.g., malaria, Kala Azar and leukaemia. A single blow may produce more than one rupture.

KIDNEY: Contusions and lacerations usually result from blunt force applied directly to the posterior or lateral aspect of the kidney as from blows to loins.

BLADDER: The bladder may be lacerated from a fall, a kick or a blow on the abdomen. When the bladder is distended, the peritoneum over the upper surface is stretched and tears, which often extends through the bladder wall and urine is extravasated into the peritoneal cavity. When the bladder is only partially distended, blunt force to the lower abdomen may cause an extraperitoneal rupture.

FEMALE GENITAL ORGANS: The uterus, ovaries or the Fallopian tubes may be contused or lacerated in severe compression injuries of the pelvis. The non-gravid uterus is not usually injured. The gravid uterus may be ruptured by a blow, kick, trampling on the abdominal wall or by instrumental criminal abortion, or in obstructed labour.

MALE GENITAL ORGANS: The penis may be injured by a squeeze or crush, and the engorged erected penis may be completely avulsed from the pubes by forceful pull. The testicles are contused from blows, kicks and squeezes. Compression or crushing of the testis may cause sudden death from cardiac inhibition.

BONES: In direct violence, fracture occurs at the site of impact and some injury to the overlying soft tissue is always present (Fig. 9-8). They may be compound or comminuted. If the violence is indirect, a simple fracture occurs in a region distant from the site of impact of the force, e.g., a fracture of the head of the radius or of the lower end of the humerus caused by a fall on the extended palm. A fall on the outstretched hand will cause Colle's fracture (fracture of the distal end of the

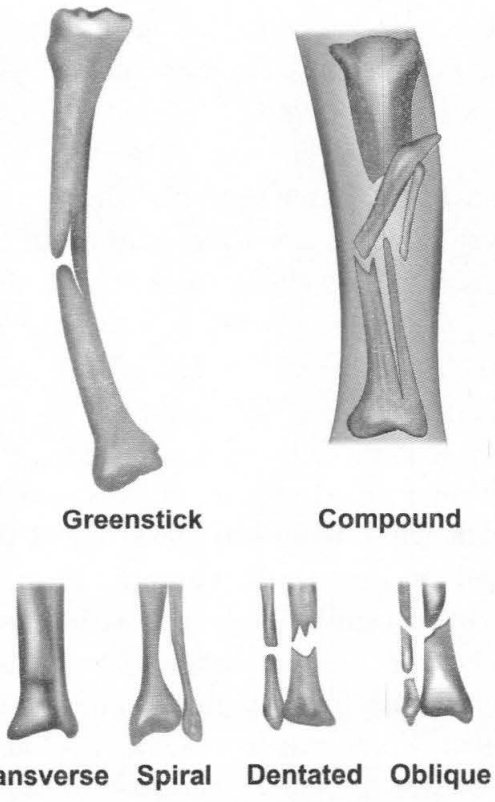


Fig. (9-8). Various types of fractures.

radius). A compound fracture from indirect violence may occur when a fragment of bone pierces the skin from the inside. In children, green-stick fractures may be more common. In childhood, slipping of an epiphysis is common, e.g., in distal end of the radius, internal epicondyle of the humerus, capitellum and distal end of the tibia. In simple closed fractures of the tibia and fibula, half to one litre of blood is usually lost and in fracture shaft of the femur about one to two litres. The calcaneum is usually fractured if the heel is struck. Other skeletal injuries, especially compression fractures of the dorsolumbar region may occur. At autopsy, a fracture may be suspected when abnormal mobility or crepitus is found. The tissues surrounding suspected fracture should be dissected to determine injuries to the soft parts.

Distinction between Antemortem and Postmortem Fractures: Fractures caused some hours before death show signs of effusion of blood, laceration of muscles and oedema, which are not seen in a fracture produced after death. A fracture produced shortly before, or shortly after death has same characters, except that in the former there may be more effused blood which penetrates further into the adjacent tissues.

TRAFFIC ACCIDENTS

(1) INJURIES TO PEDESTRIANS (FIG. 9-9): Patterns of injury:

(1) Primary impact injuries (the first part struck). (2) Secondary impact injuries (further injuries caused by the vehicle). (3) Secondary injuries (injuries caused by the victim's striking objects, such as the ground) **(Fig. 9-9)**.

In **primary impact injuries,** the part of the body involved depends upon the position of the person in relation to the vehicle when struck, i.e., whether crossing the road from one side to the other or walking with or against the traffic. The injuries depend upon the relative heights of various parts of the vehicle, i.e., bumper, fenders, radiator, door handles, etc.

Bumper injuries: In the **typical case, the victim is struck from behind by the front of the vehicle and sustains the so-called bumper injuries on legs. These injuries may be severe with fractures and extensive soft tissue damage. Usually, the tibia is fractured with forward displacement of the bone fragment, which was struck.** The fracture is usually spiral or wedge-shaped. The base of the triangular fragment of bone indicates the site of impact and

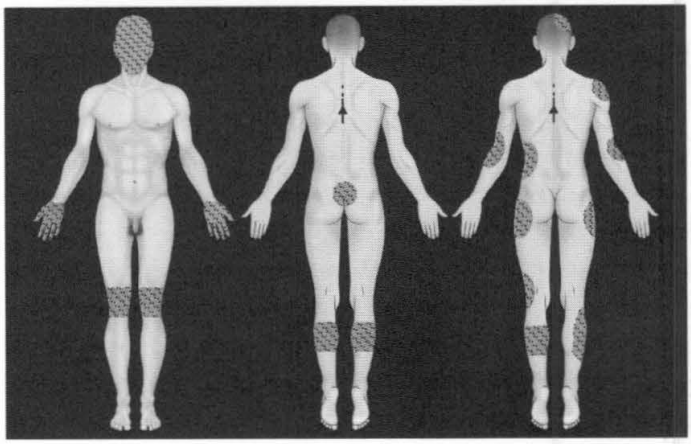


Fig. (9-9). Impact injuries; primary (cross-hatched); secondary (dotted).

the apex points in the direction in which the vehicle was travelling. If the leg is lifted, the fracture is often transverse. If the vehicle was braking violently at the moment of impact, the front end of the vehicle dips down and the legs are injured at a lower level. Frequently, bumper injuries are at different levels on the two legs or absent on one, which suggest that the victim was walking or running when struck. In children, the bumper usually produces fracture of the femur.

Other injuries: An impact against a mudguard or headlamp may result in fracture of the pelvis or fracture-dislocation of the sacro-iliac joints. If the person is facing the vehicle, he may sustain intra-abdominal injuries and injuries to the chest wall and the thoracic contents. **In primary impact injuries, the margins of the lacerations show contusions and in secondary impact injuries, abrasions.** Direct impact to the thorax may cause the rupture of the aorta below the arch at the level of the ligamentum arteriosum and sometimes laceration or rupture of the heart. Splenic lacerations cause slow oozing. Injury to the intestinal tract is less common. If the victim is struck from behind, linear superficial tears of the abdomen or the inguinal regions are seen due to overstretching of the skin. If the victim is run over by the vehicle, similar injuries may be produced due to overstretching of the skin. If the vehicle travels at high speed, deep lacerations may be produced due

to the same mechanism. Many impacts are on front corner of the car and the pedestrian may be knocked diagonally out of the path of the car. If thrown into the centre of the roadway, the person can be run over by or injured at a lower level. When a pedestrian walks into the side of a vehicle, pressure abrasions or tears on the side or front of the face are produced which are sometimes patterned. The chest or loin may be injured, sometimes with fractured ribs and rupture of lungs, diaphragm, spleen or liver. Tearing wounds may be caused by protruding objects, such as door handles, or with cuts from broken glass. Second collision refers to impact of the person against an object or outside of the vehicle.

If a **fast moving bus, truck or van having a straight and high front end** hits an adult, the impact is higher, at hip or even shoulder level. Even low speeds can cause severe injuries. If the body is hit at hip level, the victim will be pushed directly forwards in front of the vehicle. If the body is hit high up, the upper part of the body will be pushed down, or he may be carried on the radiator grille for some distance and fall in front of the vehicle as it slows down or comes to a halt, and the victim may be run over. Protruding objects may cause serious injuries.

Secondary impact injuries: If the foot is fixed, a fracture results and the buttocks and back will come in contact with the car and then pushed forward. He may sustain a fracture-dislocation of the lumbar or thoracic spine, sometimes associated with the fractures of adjacent ribs. If the feet slide forward, the whole body will fall backward with a secondary impact of the head against the windshield, or he may be thrown into the air, or to one side and strike the ground.

Secondary injuries: When he is **thrown clear of the vehicle soon after the impact, he may sustain secondary injuries which vary greatly in severity.** They may be abrasions, contusions, fractures of various bones including the skull. In pedestrians, the head is injured by direct impact with the vehicle and by striking the road. Both impacts produce fractures involving almost all parts of the skull, especially the base. Extensive surface laceration with much subarachnoid and often subdural bleeding may occur. Haemorrhage into the basal ganglia and severe laceration of the corpus callosum may occur.

Injuries due to runover: If the person is run over there may be: (1) tyre marks, (2) grazes, (3) avulsion of skin, (4) burning of the skin due to the heat produced by sliding of the body, (5) deep crushing of the internal organs, and flattening deformities of the head, chest or pelvis, (6) decapitation or complete amputation of an extremity. When a child is run over by a vehicle, fractures may not be produced, because of the elasticity of partly cartilaginous skeleton.

Rolling Injuries: Rolling injuries are produced when a vehicle with a low chassis "rolled" the victim along the roadway as it passed over him. They are mostly abrasions, grease soiling, and burns from the exhaust system. There may be fractures of the skull, neck, shoulder, shoulder girdle, ribs, vertebrae or pelvis. Patterned imprints may be present caused by parts on the undersurface of the chassis.

OCCUPANTS OF THE VEHICLES: Most of the vehicular accidents are frontal causing violent deceleration. The occupants of the vehicle will continue to move forward, even though the vehicle has stopped. The unrestrained driver first slides forward so that his legs strike the fascia parcel shelf area and his abdomen or lower chest strikes the lower edge of the steering wheel **(secondary accident)**. Then the body flexes across the steering wheel and begins to rise. The heavy head goes forward and there is flexion of cervical and thoracic spines. The head strikes the windscreen, the upper windscreen rim or the side pillar. The windscreen may break and the driver may be ejected through the broken glass on to the bonnet or on the road. While being thrown forward, the chest of the driver may strike the steering wheel, or the upper abdomen on wheel-rim. The horn boss may strike the sternum and produce a transverse fracture and crush the heart or split the aorta. The wheel rim may crush the liver, spleen or kidneys. In some cases lungs are lacerated, due to fractured ribs penetrating the lung tissue. These are often known as **'the steering-wheel impact type of injury'**. Modern design has reduced the danger of injuries by steering column by making the column telescope, hinged or collapsible. If the driver appreciates that the accident is unavoidable, he may put some of the force on his hands on the steering-wheel, and may sustain injuries to the wrists or forearms. Transmitted fracture of the femur or pelvis occurs if attempt is made to apply

the brakes. The driver and front passengers may split the head or face on a shattering windscreen frame, or may sustain a fracture-dislocation of the cervical spine due to hyperflexion of the head, or being thrown up as well as forward. When the forehead strikes the windshield, shallow penetration produces U-shaped wounds and deeper penetrations, lacerations. **Multiple punctate lacerations of the face are produced due to the shattering of the windscreen glass into multiple small fragments with relatively blunt edges ("sparrow foot" marks).** They are relatively superficial. Dislocation of atlanto-occipital joint is found in about one-third of all fatal motor vehicle accidents. In front seat passengers the pattern of injuries are almost similar to that of the driver, except steering wheel injuries.

In **front-to-side collisions**, occupants whose side was struck are usually thrown against the door interior. The head strikes the upper part of the window glass, frame and adjacent roof; upper limbs and thorax strike the window sill and adjacent door structures; and hip and abdomen strike the arm rest area.

Whiplash injury is due to a violent acceleration or deceleration force applied to the passenger, usually front seat occupant. When the vehicle comes to a sudden stop due to head-on obstruction, the heavy head continues to move forward (acute hyperflexion). When the body comes to rest, there is then a reactionary hyperextension as the head is thrown backwards, this double movement being known as the 'whiplash'. If a vehicle is hit violently from behind by another vehicle, then the head is thrown violently backwards into hyperextension and then comes forward after the body stops movement. In either case, this **violent extension-flexion movement can cause dislocation of the upper cervical spine, or less commonly a fracture-dislocation in the lower part of the spine at about C 5-6.** Fatal contusion or laceration of the spinal cord may occur without fracture of spine.

Back Seat Occupants: During violent deceleration, unrestrained back seat occupants are projected forwards and strike the back of the front seats including head rests. They may be thrown over the seats and fall on the front seat passengers or sometimes ejected through the windscreen.

Sometimes, the door may spring open and the person may fall out **(occupant ejection)**. When the car overturns, the occupants may be crushed, showing classical traumatic asphyxia, or the chest and abdomen may be crushed with serious injury to the heart, lungs, liver, spleen, or kidneys. Depending on the circumstances of the accident, the occupants are burned, asphyxiated or drowned whilst trapped in the interior of a car.

Tail-gating occasionally occurs with cars driving into the back of large trucks. In such case, the windscreen and front of the passenger compartment are smashed with severe injuries to the occupants of the front seat.

Seat Belt Injuries: Seat belts are of the lap strap and shoulder diagonal type. The belts hold the occupants back against the seat and prevent forward movement against steering wheel and windshield, and ejection from the vehicle. Small intestine avulsions, intestinal, omental, mesenteric lacerations and intestinal contusions and perforations may occur, known as **"seat belt syndrome"**, due to acute flexion over a lap strap. Rupture of the spleen, liver, pancreas, caecum and bladder may occur due to compression between belt and vertebrae. Abrasions, contusions and haematomas of the lower abdomen can occur.

Triage is the sorting out and classification of casualties of war or other disaster, to determine priority of need and proper place of treatment.

(2) CYCLISTS AND MOTOR CYCLISTS: This includes conventional motor cycles, motor scooters, mopeds and pedal cycles. Accidents are commonly caused by turning in front of a vehicle from one side or the other. The cycle is hit, and the person is thrown violently to the ground or into some other object or on to the car. In being thrown, they may injure the groins or legs due to protruding objects, like handlebars, levers or mirrors, long tears being common. The handlebar may tear the liver or the spleen and cause massive internal bleeding. The head or shoulder may hit some object first, or they may be thrown beneath a vehicle and crushed like any pedestrian. Injuries to legs may occur in relatively low speed accidents due to the machine falling heavily on them. Falling from the vehicle, especially at speed can cause visceral damage, especially rupture of the liver and spleen. **Crash helmet reduces friction of the**

head against the ground and makes deceleration less drastic, by allowing the protected head to skid across the ground. Helmets reduce the risk of head injury by about 30%, and fatalities by about 40%. When a crash helmet is worn, the crown may be protected, but the whole head may be "eggshelled" on to the base or cervical spine with fracture of the spine. Occasionally, when the helmet is pinned or crushed, the chin strap may be drawn upwards and cause strangulation. About 50% of helmetless motor cyclists sustain head injury. The classical fatal injury in both motor cyclists and pillion passengers is fracture of the skull, usually from secondary impact with ground. Temperoparietal fractures are common often with contrecoup brain injury. In many types of road accidents, a fracture is produced across the floor of each middle fossa passing through the pituitary fossa, often associated with fissured fractures passing upwards into the temporal bones. At autopsy, the **base of the skull is seen divided into two halves, each moving independently of each other like a hinge, the so-called motor cyclist's fracture.**

Bicycle spoke injury occurs when a rider or passenger on a bicycle slips from the seat and the foot or leg passes through the spokes of the wheel. In this soft tissue is crushed with internal avulsion or damage.

Tail gating: The motor cyclist may drive into the back of a large vehicle, e.g., truck, known as **"underrunning" or "tail-gating"**. This may occur due to the sudden and unexpected stoppage of the truck or when the motor cyclist is at high speed in darkness. In such cases, the motor cyclist's head and shoulders are smashed against the tail-board and in some cases decapitation occurs.

BOXING INJURIES: A wide range of injuries are produced, but the head is frequently injured. Fracture of skull is rare but subdural haemorrhage occurs in about one-third of fatal cases. **Some victims may show pontine haemorrhage, the so-called "boxer's haemorrhage".** Bleeding into the midbrain is seen. Retinal detachment may occur. Deterioration in speed and coordination are the chief symptoms of the onset of **punchdrunk** (traumatic encephalopathy or dementia pugilistica) condition. The mechanism is not known, but probably repeated blows to the head produce small haemorrhages and degenerative changes in brain. In extreme cases, the punchdrunk is easily recognised from his slurred speech, defective memory, slow thought process, stiff limbs and expressionless face, outbursts of violence and finally dementia.

RAILWAY INJURIES

When the person walks on the track, primary injuries are seen on the front or the back, due to contact with the protruding parts of the engine. Secondary injuries are produced due to being thrown or run over which are soiled with oil and dust (Plate 12-67). When the person is crossing the line, primary injuries are seen on the side of the head and shoulders. If the person is bending down, the primary injuries are seen on the back and the buttocks, and secondary injuries on the face and front of the body. When the person walks at the side of the line, the shoulder or head may be hit by some protruding part of the train. When a person falls from the train, multiple injuries are produced. When a person leans out of the window, head injuries are produced due to railway fixtures, bridge abutments or tunnel sides, etc., and person may be found in the carriage or on the side of the track. A person trying to board a fast moving train may be thrown off or may get crushed between train and the platform. Accidents may also occur during shunting. When a person is run over by a train, very severe mutilation of the body may occur with separation of the limbs and protrusion of organs. Wheel marks, dirt and grease contamination may be seen on the body. In collisions, and derailment, fractures of the legs and spine are produced. Head injuries, contusions, cuts and burns are also produced. Most injuries are produced due to crushing and trapping within distorted carriages.

Sometimes, a person is killed and the body placed on the railway track to simulate suicide or accident. Examination of the scene of the crime and the body for marks of violence will often solve the problem.

CHAPTER

10 Medicolegal Aspects of Wounds

Homicide: (I) Lawful: (1) Justifiable. (2) Excusable. These terms are not used in Indian law. **(II) Unlawful:** (1) Murder. (2) Culpable homicide: (a) amounting to murder, (b) not amounting to murder. (3) Rash or negligent homicide.

Justifiable Homicide: This is the homicide which is **justified in the circumstances which led to the killing of a person.** This may occur (1) in the administration of justice, like execution of sentence of death, (2) the maintenance of justice, e.g., in suppressing riots or executing arrest or killing in course of violent crime, e.g., a woman who kills a person who attempts to rape her.

Excusable Homicide: This is homicide **caused unintentionally by an act done in good faith.** This includes (1) killing in self-defence when attacked, provided there is no other means of defence, (2) causing death by accident or misadventure, (3) death following a lawful operation, (4) homicide committed by an insane person.

INJURY: Any harm, illegally caused to any person in body, mind, reputation or property (S. 44, I.P.C.).

CULPABLE HOMICIDE: Culpable homicide is causing death by doing an act (1) with the intention of causing death, or (2) with the intention of causing such bodily injury as is likely to cause death, or (3) with the knowledge that such act is likely to cause death **(S. 299, I.P.C.).**

Explanations: (1) A person who causes bodily injury to another who is suffering from a disorder, disease or bodily infirmity which accelerates the death of the person, shall be deemed to have caused his death. (2) Where death is caused by bodily injury, the person who caused such bodily injury shall be deemed to have caused death, although by using proper remedies and skilful treatment, the death might have been prevented. (3) The causing of the death of a child in the mother's womb is not homicide.

MURDER: Culpable homicide is murder (1) if the act by which the death is caused is done with the intention of causing death, or (2) if it is done with the intention of causing such bodily injury as is likely to cause death, or (3) if it is done with the intention of **causing bodily injury which is sufficient in the ordinary course of nature to cause death,** or (4) if the person committing the act knows that it is so dangerous that it must in all probability cause death or is likely to cause death **(S. 300, I.P.C.).**

Exceptions: Culpable homicide does not amount to murder, if the act by which it is caused is done: (1) under grave and sudden provocation, (2) in good faith of the right of private defence of person or property **(S. 96 to 106, I.P.C.),** (3) for advancement of public justice, (4) without premeditation, and (5) when person above the age of eighteen years takes the risk of death with his own consent **(S. 87, I.P.C.).**

A bodily injury which is likely to cause death is one, in which death is not only possible, but is likely. It constitutes a grave danger to the life of the affected person due to its severity or of the structures involved. It applies in special cases where person injured is in such a condition or state of health, that his or her death is likely to be caused by an injury which would not ordinarily cause the death of a person in sound health, and here the person inflicting the injury knows that owing to such condition or state of health, it is likely to cause the death of the person injured. **A bodily injury which is sufficient in the ordinary course of nature to cause death is one, as a result of which death is very probable.** Some

examples are: stab injuries or rupture of the heart; injury to the large blood vessels; stab on the abdomen, chest or other parts of the body injuring an internal organ or a large blood vessel; hurt causing rupture of spleen, liver or an internal organ; severe injuries of the brain and spinal cord; incised wound on the neck; compound fracture of the skull; squeezing of the testicles; penetrating wounds or rupture of the gastrointestinal canal; extensive burns and scalds; hanging or strangulating a person; smothering or gagging; drowning; failure to tie the cord after it is cut in a newborn; administering large dose of poison, etc. A number of injuries, none of which is sufficient in itself to cause death may together by their cumulative effect be sufficient to cause death. **The offence is culpable homicide, if the body injury intended to be inflicted is likely to cause death. It is murder, if such injury is sufficient in the ordinary course of nature to cause death. The distinction is fine but appreciable. The nature of the weapon has to be considered**. A blow from the fist or a stick on a vital part may be likely to cause death; a wound from a knife in a vital part is sufficient in the ordinary course of nature to cause death.

Punishment for murder: Death, or imprisonment for life, and also fine **(Sec. 302, I.P.C.)**.

Punishment for culpable homicide not amounting to murder: Imprisonment for life, or up to ten years, and also fine **(Sec. 304, I.P.C.)**.

Causing death by negligence: Whoever causes the death of any person by doing any rash or negligent act not amounting to culpable homicide shall be punished with imprisonment for a term which may extend to two years or with fine, or with both **(S. 304–A, I.P.C.)**.

DOWRY DEATHS

S. 304-B, I.P.C.: Where the **death of a woman** is caused by any burns or bodily injury, or occurs under abnormal circumstances **within seven years of her marriage, and it is shown that soon before her death she was subjected to cruelty or harassment by her husband or any relative of her husband,** for or in connection with, any demand for dowry, such death shall be called "dowry death", and such husband or relative shall be deemed to have caused her death. The punishment shall be imprisonment of not less than seven years, but may extend to life imprisonment.

S. 498–A, I.P.C.: Whoever being the husband or the relative of the husband of a woman, subjects such woman to cruelty shall be punished with imprisonment up to three years.

Dowry deaths occur either by murder of a married woman, or she herself committing suicide being unable to bear harassment or cruelty for not fulfilling the promises by her parents or her relatives or of those interested in her marriage. Such murders are invariably committed secretly, either in the house or at a place where outsiders may not witness it. The bride may be burnt or killed by various methods. Domestic discord, often in families with numerous children, drunkenness and demand for dowry are usually responsible. The usual defence in all dowry death cases is that either the woman committed suicide, or death occurred accidentally due to burns while cooking food.

The doctor should make a record of the history of the injuries and the persons responsible for it, when the victim is brought to the hospital, noting the time and the name of the person who gave the history. The condition of the victim should be recorded at frequent intervals. Care should be taken that no person has discussion with the victim, before doctor records the statement of the victim to avoid any undue influence being exerted on her. A Magistrate may be called to record the dying declaration.

Inquest should be conducted by a Magistrate or police officer not below the rank of Deputy Superintendent of Police, and **autopsy should be carried out by two doctors.**

Wife Battering: A battered wife is a woman who has received deliberate, severe and repeated demonstrable physical injuries from her husband. The involved men below the age of 40 suffer from personality or neurotic disorders, while those over 40 suffer from psychiatric disorder.

SUICIDE: Attempt to commit suicide is punishable with imprisonment up to one year **(Sec. 309, I.P.C.)**, and **abetment of suicide** is punishable with imprisonment up to ten years **(Sec. 306, I.P.C.)**.

HURT: Hurt means bodily pain, disease or infirmity caused to any person **(S. 319, I.P.C.)**.

GRIEVOUS HURT: According to **S. 320, I.P.C.** any of the following injuries are grievous. (1) Emasculation (loss of potency). (2) Permanent privation (loss) of

sight of either eye. (3) Permanent privation of hearing of either ear. (4) Privation of any member or joint. (5) Destruction or permanent impairing of the power of any member or joint. (6) Permanent disfiguration of the head or face. (7) Fracture or dislocation of a bone or tooth. (8) Any hurt which endangers life, or which causes the victim to be in severe bodily pain, or unable to follow his ordinary pursuits for a period of 20 days. Ordinary pursuits include acts of daily routine of a person, such as eating food, taking bath, going to the toilet, etc., but do not refer to the duties that constitute the job of a person.

Simple Injury: Simple injury is one which is neither extensive nor serious and which heals rapidly.

Punishment for voluntarily causing hurt: Imprisonment up to one year, or with fine or both **(S. 323, I.P.C.)**.

Punishment for voluntarily causing grievous hurt: Imprisonment up to seven years and also fine **(S. 325, I.P.C.)**.

Dangerous Weapons or Means: According to **S. 324 and 326, I.P.C**. dangerous weapons or means include any instrument for shooting, stabbing or cutting, or any instrument, which used as a weapon of offence, is likely to cause death; fire or any heated substance; poison or any corrosive substance; explosive substance; any substance which is harmful to the human body to inhale, to swallow, or to receive into the blood, or by means of any animal.

ASSAULT: An assault is an offer or threat or attempt to apply force to the body of another in a hostile manner **(S. 351, I.P.C.)**.

TORTURE

The World Medical Association (Declaration of Tokyo, 1975) defined torture in relation to detention and imprisonment as **"The deliberate, systematic or wanton infliction of physical or mental suffering by one or more persons** acting alone or on the orders of any authority, to force another person to yield information, to make a confession or for any other reason".

Torture may be carried out by (1) Criminal and terrorist groups, and (2) By the police or other security force personnel during the detention and interrogation of prisoners and suspects.

Objects: (1) To obtain information, if a person is suspected to have committed a crime or indulged in antinational or terrorist activities. (2) To obtain testimony incriminating others. (3) To sign a document confessing a crime. (4) To take revenge against a person or his family members, by rape, kidnapping, etc. (5) To spread terror in the community by militant groups or by dictators ruling the country. (6) To destroy the personality of individuals who raise their voices against dictatorial rule, or oppression in the society.

METHODS: (I) PHYSICAL ABUSE: (1) Beating is the most common form. Fist, foot or an instrument, such as *lathi,* metal or wooden bars, whips, belts, cycle chains, etc., are used. (2) Falanga (falaka, bastinado): In this canes or rods are used to beat on the soles of the feet. (3) Telefono: It consists of repeated slapping of the sides of the head (over ears) by the open palms of the assailant. (4) Beating on the abdomen while lying on a table with the upper half of the body unsupported. (5) Beating the head. (6) Fingers are twisted or pencils, etc., are kept between two fingers and squeezed hard. (7) Making the victim chew hard on pieces of metals, stones, etc., or pulling teeth by clips or forceps. (8) Mutilation: Chopping of ears, nose, fingers, etc. (9) Disfiguration by throwing corrosives on face or other parts of body. (10) Use of continuous high-pitched sound. (11) Forced immersion of head (wet submarine). (12) Tying of a plastic bag over the head (dry submarine). (13) Suspension of body by the wrists. (14) Suspension of body by the arms or neck. (15) Suspension by the ankles. (16) Head down from a horizontal pole placed under the knees with the wrists bound to the ankle. (17) The victim is asked to stand in hot sun on one leg for prolonged periods. (18) Forced straddling of a bar (saw horse): Perineal or scrotal haematoma occurs. (19) Burns caused by cigarette butts is common (Plate 16-93 and 16-94). Other methods are application of heated solid body, molten rubber dripped on skin, kerosene soaked rags wrapped around limbs and set fire, hot iron, throwing boiling water, etc. (20) Cold: Victim is made to lie on a cold and damp floor or on an ice slab without clothing. (21) Electric shock (cattle prod). (22) Heated metal skewer inserted into the anus (black slave): Perianal or rectal burns. (23) Dehydration. (24) Animal bites.

Some methods of physical abuse may not leave any apparent marks of violence, such as hair-pulling, face slapping and blows to the side of the head which may cause perforation of the eardrum. Blows to the abdomen may not cause any apparent mark. Pinching, squeezing and blows to the testes may produce bruising of the skin

and swelling and tenderness of testes. The soles may be struck with a baton, ruler, etc.

(II) MENTAL TORTURE: (1) Solitary confinement in a dark place. (2) Blindfolding for a long time, or frequent transfer from one place to another blindfolded. (3) Starving the victim. (4) Causing mental anguish by giving false information to victim regarding tragedy involving wife and children.

(III) SEXUAL TORTURE: (1) Infliction of injuries to private parts or introducing foreign bodies into the rectum or vagina, or mutilation of breasts or genitals. (2) Raping the victim or undressing before others, or sexually tortured by trained animals, etc.

(IV) PHARMACOLOGICAL TORTURE:Various drugs, such as drugs to induce self-disclosure, muscle relaxants, pain-inducing drugs, or psycho-pharmacological drugs, etc.

DEATH IN CUSTODY

(I) DURING ARREST: (1) Traumatic asphyxia may occur if the offender resists arrest, and a number of policemen fall upon him to overpower him. (2) If there is a struggle, blunt injuries sustained by the offender, by the use of fist, arm or leg, riot stick, etc., by the police, or from head injury from falls against ground, wall, etc., may cause death. (3) In CHOKE HOLDS forearm is placed straight across the front of the neck (Fig. 10-1). The free hand grips the wrist and pulls it back, which causes obstruction of the airway and carotid arteries and immediate unconsciousness. Rarely, death may occur due to hypoxia and release of catecholamines due to struggle, resulting in fatal cardiac arrhythmia. (4) In the CAROTID SLEEPER HOLD, the arm is placed about the neck with the antecubital fossa centered at the midline of the neck. The free hand grips the wrist and the arm is pulled backwards, which compresses the carotid arteries and jugular veins but not the trachea (Fig. 10-2). Consciousness is lost in 10 to 15 seconds. Rarely, death may occur due to hypoxia and release of catecholamines.

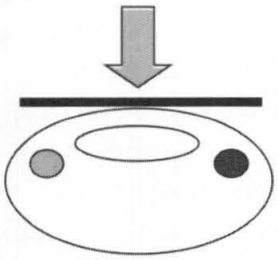


Fig. (10-1). Carotid hold. Force applied across the front of neck compressing the airway.

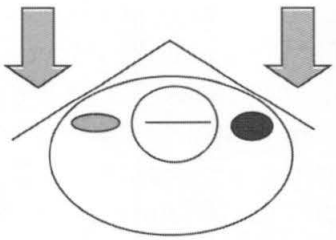


Fig. (10-2). Carotid sleeper. Direction of force on neck aimed at compressing the carotid arteries.

(II) DURING LOCKUP: (1) If the offender was severely intoxicated, he may die of acute alcoholic poisoning, or he may aspirate the vomit and die of choking. (2) The drunken offender may fall on a hard surface on the occiput. If on autopsy contrecoup injuries are found, it indicates deceleration injury rather than an assault with a weapon. Physical injuries may also occur from falls in drug addicts used to amphetamine, cocaine or hallucinogens. (3) Torture by kicking and stomping, or a backward blow from the point of an elbow on the face, neck, or abdomen, fist blows, etc., inflicted with sufficient force, can cause severe damage and death.

Suicide in custody is common. The common method is hanging. Sometimes a poison may be consumed, or he may jump from a height, or cut his throat or a large blood vessel.

Death may occur due to natural causes, usually cardiovascular. The emotional upset may precipitate an acute cardiac crisis in the presence of severe preexisting disease.

WOUND CERTIFICATE

Medicolegal injury cases should be examined without delay at any time of day or night. All details of examination of injured person, whether admitted into hospital or treated as out-patient have to be entered in an **Accident Register,** maintained in all Government Hospitals. This register is a confidential record and should be in safe custody of the medical officer. It has to be produced in the Court if asked for. A detailed and accurate record should be made by the doctor at the time of the examination. The doctor is required to fill in a printed form of injury or wound certificate, one copy of which is sent to the investigating police officer in a sealed cover, and the other is retained for future reference. The doctor should inform the local police station over telephone or by a letter of intimation.

Preliminary particulars: The following particulars should be noted: (1) Serial number. (2) Name, age and sex of the injured person and address. (3) Father's or guardian's name. (4) Date, time and place of examination. (5) Name and number of the accompanying police constable and the police station to which he belongs. (6) Names of the persons who had accompanied the injured person with their address. (7) Two identification marks. (8) The size of the victim, i.e., stature, body weight and development. (9) A brief statement of the injured person as to how he was injured. The consent of the person for examination should be taken. The following are the various entries in the wound certificate.

(1) Nature of each injury: All injuries observed, however insignificant they may appear, should be noted. The nature of each injury, i.e., abrasion, contusion, incised wound, lacerated wound, stab wound, burns, fracture, dislocation, etc., should be noted. It is better to group them anatomically, e.g., injuries of the head, of the trunk or of a limb. **A lens should be used to get an accurate idea of the nature of the edges, ends** and floor of a wound. The presence of any foreign material in the wound, e.g., metal, glass, hair, dirt, etc., should be noted.

(2) Size, shape and direction of each injury: All injuries should be measured with a tape and never guessed, and the amount of blood extravasated should be measured. The shape of the wound, e.g., circular, oval, triangular, elliptical, etc., bevelling of the edges should be noted. The direction of the wound, i.e., horizontal, vertical, oblique, etc. should be noted with regard to the anatomical position of the body. In cases of stab and firearm wounds, direction of the wound should be noted.

(3) On what part of body inflicted: The exact situation of wound with reference to some anatomical landmark, e.g., the middle line, a bony structure, a joint, the navel or the nipple should be mentioned. Technical terms should be avoided as far as possible.

(4) Simple, Grievous or Dangerous Injury: Against each injury it should be noted whether it is simple or grievous. **The injured person must be kept under observation, if the nature of a particular injury cannot be made out at the time of examination,** e.g., in head injury where the symptoms are obscure, or abdominal injuries with vague symptoms. In all injuries **when a fracture of a bone is suspected, an X-ray examination should be done for confirmation**. In such cases, the report should not be given hastily, if there is pressure from the police.

(5) By what weapon inflicted: In many cases, the examination of the wound and clothes gives a fairly definite information about the kind of weapon. With stabs and incised wounds, there is not much difficulty; wounds caused by broken glass may simulate either, but irregularity of the edges will be found in some parts of the wound, and if glass pieces are found it clears all doubts. Wounds made by blunt instruments, blows or kicks over bony prominences may simulate incised wounds, but an examination with hand lens helps the diagnosis.

A **foreign body** found in a wound, e.g., a piece of glass, a splinter of wood, the broken off point of knife or the bullet, shots or wadding of a firearm, may indicate the manner in which the wound was inflicted, and may even help to identify the weapon with which the injury was caused.

The **clothes should be examined for the presence of cuts, tears or burns, and it should be seen if these injuries correspond to the injuries on the body.** If the clothing was very loose and was disarranged during the struggle or folded on itself, these may not correspond with the wounds.

(6) Whether the weapon was dangerous or not? A dangerous weapon is any instrument used for shooting, stabbing or cutting or any instrument which used as a weapon of offence is likely to cause death.

(7) Remarks: The general condition of the patient should be noted under this column, such as conscious or unconscious, bleeding from the ears, nostrils, mouth, etc., paralysis, state of shock, etc. **The age of the injury should be noted.** It is only possible to give an approximate time. The age of the injury is very important, for its appearance may or may not correspond to the time when it is alleged to have been produced, and all the injuries found on a person may not have been produced on the same day.

When a victim of suicide, homicide or accident dies in the hospital, the medical officer should report the matter to the police immediately. **When a dead body is brought to the hospital, do not examine the injuries.**

CAUSES OF DEATH FROM WOUNDS

The causes of death from the wounds are: (1) Immediate or direct. (2) Remote or indirect.

(A) IMMEDIATE CAUSES: (1) Haemorrhage: Haemorrhage means escape of blood from the cardiovascular system. The haemorrhage may occur at the time of injury (primary haemorrhage). **Secondary haemorrhage** occurs from the same site as primary haemorrhage, but is usually delayed for several hours (up to 24 hours or more) after the injury. The sudden loss of blood is more dangerous than the same quantity lost slowly. The rapid loss of one-third of the blood

of the body causes death. Children, women and old people die from the loss of much smaller quantity. A minor injury may produce death from haemorrhage in persons with haemorrhagic diathesis or haemophilia. An approximate idea of loss of blood may be had from the soaking of clothes and the surroundings of the victim and from the anaemic state of the viscera.

(2) Reflex Vagal Inhibition: In this, there may not be visible external injury.

(3) SHOCK: Shock is a circulatory disturbance characterised by an acute persisting reduction of blood flow in the peripheral vascular bed and an inadequate perfusion of the tissues.

Primary Shock: Primary or neurogenic shock (vaso-vagal shock or reflex cardiac arrest) results from a sudden reduction of venous return to the heart due to neurogenic vasodilation with pooling of blood in the peripheral vascular bed, especially in the splanchnic area. It may follow any form of stress. Psychological factors, such as fear, grief, anxiety, emotion and pain due to various causes also play a large part. It may occur when a few ml. of blood are withdrawn, or from the sight of blood or the anticipation of an injection.

Secondary Shock: It is more serious and may be fatal. There is progressive circulatory failure and damage to the tissues. **It is called secondary or delayed shock because the circulatory disturbance usually develops gradually or after a latent period following the injury.**

Types: Shock can be classified on the basis of the stressful circumstances under which it occurs. **(1) Haemorrhagic shock:** This occurs when the blood loss is sufficient to impair peripheral perfusion, which produce a sufficient degree of systemic anoxia. Loss of 10 to 40 percent of the total blood volume produces shock. **(2) Traumatic or wound shock:** This is the most common type of shock. The blood volume may be reduced due to haemorrhage from ruptured vessels, an excessive loss of plasma from damaged capillaries or venules at the site of injury, or a combination of both factors. **(3) Burn shock:** It results from loss of plasma, absorption of necrotic tissue, and bacterial sepsis following extensive burns. **(4) Surgical shock:** It occurs either during or immediately after surgical procedures. It may be due to a combination of causes: anaesthesia, loss of blood and plasma, emotional reactions, infections and probably liberation of substances from severely traumatised tissues that may influence the circulation. **(5) Cardiac or cardiogenic shock:** It results from the decreased cardiac output, reduced blood flow and deficient delivery of oxygen to the tissues, following injury to the pumping mechanism. **(6) Septic shock:** It develops in the presence of severe infection. Loss of fluid from the circulation (generalised increased vascular permeability), or pooling of the blood in the peripheral vascular bed (vasodilation), cause circulatory failure. **(7) Endotoxic shock:** This is closely related to, if not identical to septic shock. It is seen in patients having Gram-negative infections. Bacterial endotoxins induce intravascular coagulation prior to endothelial damage.

(4) Mechanical Injury to a Vital Organ: Severe injury to a vital organ, such as crushing of brain, heart, etc. is rapidly fatal. Death may occur from a slight injury inflicted on a previous diseased organ, e.g., rupture of enlarged spleen, perforation of a chronic intestinal ulcer, bursting of an aneurysm, etc.

(B) REMOTE CAUSES: Though the injury does not directly cause death, the victim may die after a varying period from remote causes. **Even when the victim dies of complications, an assailant is directly responsible, if death can be traced to likely result of the injury.** The assailant is responsible even if the victim was in a bad state of health when attacked, and if the injury inflicted by the accused accelerated or precipitated the death.

(1) Infection: Wound infection may be caused by: (1) Organisms normally present on the body surfaces, e.g., streptococci, Staphylococci, B. pyocyaneus, Coliform bacilli, Friedlander's bacilli, and B. welchii. (b) Organisms invading the tissues from the environment, e.g., streptococci, staphylococci, C. diphtheriae, Coliform organisms, Cl. welchii and Cl. tetani. Primary wound infection is caused by organisms, which are carried into the wound at the time of the injury, e.g., from the skin, clothing, dirt, etc. Secondary wound infection is caused by organisms which invade the wound after injury, e.g., by airborne droplet infection, contaminated dressings, etc.

(2) Gangrene or Necrosis: It results from severe crushing of part and tearing of blood vessels.

(3) Renal failure following trauma: (Acute tubular necrosis: Crush Syndrome): Traumatic tubular necrosis may occur in cases of trauma to the limbs in which necrosis of the muscles occurs, which may be due to ischaemia of direct compression. It results from severe crushing of the muscles, especially those that involve the lower limbs, e.g., under fallen masonry, industrial and vehicular accidents, etc. In traffic accidents, necrosis of muscle may occur without any prolonged crushing injury. The casts may be due to reduced filtration rather than tubular damage. Death usually occurs in one to two weeks after the injury. The cause is obscure, but may involve disturbance of renal blood flow and ischaemia. There are no specific gross changes in the kidney.

(4) Neglect of Injured Person: Death may occur from complications arising from a simple injury due to bad treatment, or negligence on the part of the doctor, or to negligence or wilful disobedience on the part of the patient.

(5) Surgical Operation: The assaulted person is not bound to submit himself for operation. If death occurs due to this omission, the assailant becomes responsible. If death follows a surgical operation performed for the treatment of an injury, the assailant is responsible for the result, if it is proved that the victim would have died even without the operation, and that the operation was thought necessary, and was performed by a competent surgeon with reasonable care and skill. When anaesthesia is given to an injured person before operation, and if it is proved that the patient would not have died but for its administration, the person causing injury is liable to be punished for culpable homicide. **If hurt or wound is not fatal, and if it is clearly proved that death of the victim is caused by application of harmful medicines by himself or those about him, this cannot be regarded as murder.**

(6) Natural Disease: Some natural disease may be present which was the actual cause of death, but in which death was accelerated by assault, e.g., persons suffering from miliary aneurysms, fatty degeneration of the heart, etc., may die due to slight violence, which may be a precipitating cause.

(7) Supervention of Disease from a Traumatic Lesion: Fibrous scar tissue formed due to healing may contract and produce complications, e.g., fibrous scar in a hollow muscular organ may produce stricture and obstruction. If a fibrous scar is under continuous tension, it may stretch and produce complication, e.g. a fibrous scar in the wall of an artery may bulge into a traumatic aneurysm. Damage of an artery by a bullet may produce false aneurysm, which may rupture later. Direct injury to the coronary artery by thoracic trauma may be followed by coronary thrombosis. A wound of the abdominal wall, after healing may be followed by strangulated hernia with fatal result. An injury affecting lower portion of spinal cord may cause paraplegia which may cause death from septic cystitis or bed sores and general exhaustion.

(8) Thrombosis and Embolism: The most common sites of thrombosis are in deep femoral, the popliteal and posterior tibial veins, and occur usually in traumatic lesions of lower extremity, especially fractures of long bones. The thrombus usually develops in ten to twenty days, and is detached in part or whole and travels to pulmonary artery. The embolus which blocks the pulmonary trunk causes death in few minutes due to vagal shock, acute asphyxia or right-sided heart failure, and no infarction is found in the lungs. Arterial thrombosis and embolism is rare. Bullet wounds and stab wounds of the carotid arteries may injure the intima and a thrombus may develop at the site of the injury, followed by embolus of middle cerebral arteries.

(9) Fat Embolism: Fat embolism is caused by: (1) fracture of long bones, (2) by an injury to adipose tissue which forces liquid fat into the damaged blood vessels, (3) by injecting oil into circulation, e.g., in criminal abortion, (4) occasionally due to natural disease without trauma as in sickle cell anaemia.

The presence of fat droplets in the bloodstream indicates that the injury was produced during life, except in case of burning and advanced putrefaction. The emboli enter the pulmonary vessels and obstruct the flow of blood through the lungs. The diagnosis is made by microscopic examination of frozen section of the lungs stained for fat with Sudan III, Scharlach R or osmic acid. The pulmonary vessels, arterioles and capillaries are seen filled with globular fat emboli. The

lungs show congestion, oedema and slight hypostatic pneumonia. Death usually occurs about the tenth day from asphyxia due to impairment of gaseous exchange in the lungs, but may be delayed up to three weeks. The brain, heart and kidneys may show few scattered emboli. Cerebral fat embolism develops when the fat emboli are forced through the pulmonary capillaries into the systemic circulation in sufficient quantity to affect the brain.

(10) AIR EMBOLISM: The causes of pulmonary air embolism are: (1) Incised wounds of the lower cervical or clavicular regions involving the jugular or subclavian veins. (2) A wound of the superior longitudinal sinus inside the skull. (3) Crush injuries of chest. (4) Faulty technique in giving intravenous injection with the gravity apparatus. (5) Artificial pneumothorax and pneumoperitoneum. (6) Oral-genital sex in a pregnant woman. (7) Injection of air under pressure in Fallopian tube, to test its patency. (8) Caesarean section, version or manual extraction of the placenta may introduce air into uterus. (9) Injection of air or fluid mixed with air or soap water into the pregnant uterus for procuring abortion. (10) Caisson's disease.

The air is carried in the blood stream to the right heart, where it is churned into a frothy mixture, which is driven into branches of the pulmonary artery and lung capillaries. **About 100 ml. of air introduced under pressure is necessary to produce fatal pulmonary air embolism.** Air in the coronary arteries indicate systemic embolism. In air embolism, right ventricle is distended with air under pressure, and bright-red frothy blood is found in the right side of the heart, vena cavae, pulmonary arteries and coronary veins. The blood is fluid throughout the body, the viscera are congested, and petechiae are present in the serous surface and in the white matter of the brain. In artificial respiration in a dying person right ventricle may contain frothy blood. Death from air embolism commonly occurs within a few minutes and is usually not delayed beyond 45 minutes.

Systemic air embolism occurs when air in sufficient quantity enters a vein of the pulmonary system, and is carried through the left side of the heart to block arterioles and capillaries in different parts of the body, but especially in the brain and the heart. Penetrating wounds of the chest are the common cause, e.g., puncture wound in a lung which does not collapse may penetrate a vein and a bronchiole and allow air to enter the vein. Crushing injuries of the chest and major or minor surgical procedures upon the thorax also produce air embolism. One to two ml. of air may be enough to produce death, because the air travels directly from lungs to the left side of the heart, from which it can be forced into the coronary or the cerebral arteries.

(11) ADULT RESPIRATORY DISTRESS SYNDROME (ARDS): It occurs due to heavy impacts on the thorax, blast injuries to the chest, infections, toxins, systemic shock, irritant gases, aspiration of gastric contents, near drowning, etc. in which there may be "diffuse alveolar damage". The victim may suffer from severe dyspnoea and progressive respiratory failure due to poor exchange of gases. The lungs become stiff, heavy and oedematous.

(12) DISSEMINATED INTRAVASCULAR COAGULATION (DIC): It occurs due to trauma, infection and other acute events. It is a consumption coagulopathy associated with blood clotting mechanism. There is an abnormal activation of the coagulation process within the blood vessels. Fibrin is consumed and precipitated in vessels, causing both vascular obstructive effects and a haemorrhagic diathesis from depletion of the coagulative system. The complications are microvascular obstruction leading to infarction and bleeding.

MEDICOLEGAL QUESTIONS ON TRAUMA

(1) Can postmortem injuries be distinguished from antemortem injuries? (Table 10-1)

Injuries inflicted shortly before death cannot be distinguished from those inflicted shortly after death. The physical components of vital reaction are: swelling, effusion of lymph, leucocytic infiltration, pus formation or evidence of repair. White blood cells are capable of movement for few hours after cardiac arrest. This makes a "vital reaction" to injury of doubtful reliability. In trauma to the living tissue, two zones are seen around the wound: (1) Close to the edge of the wound, there is decreasing enzyme activity, possibly as early sign of imminent necrosis. (2) A deep peripheral zone which shows an increase in enzyme activity possibly a defence mechanism (Fig. 10-3).

Within a few minutes of the injury, dilatation of the capillaries and migration of leukocytes may be seen. Emigration of leucocytes occurs within an hour. Monocytes appear in the exudate after twelve hours. In aseptic inflammation, the maximum exudate is found within 48 hours. In an infected wound pus may be seen after 36 hours. Reactive changes in the tissue histiocytes and swelling of the vascular endothelium may be seen within an hour of injury. At the site of injury, the fibroblasts show reactive changes

Table (10-1). Difference between antemortem and postmortem wounds

Trait	Antemortem wound	Postmortem wound
(1) Margins:	The edges are swollen, everted, retract and wound gapes.	Edges do not gape but are closely approximated.
(2) Haemorrhage:	Abundant and usually arterial.	Slight or more and venous.
(3) Spurting:	Signs of spurting of arterial blood on the body, clothing or in its vicinity present.	No spurting of blood.
(4) Extravasation:	Staining of the edges of the wound and extravasation in neighbouring subcutaneous and interstitial tissues which cannot be removed by washing.	Edges and cellular tissues are not deeply stained.
(5) Coagulation:	Firmly coagulated blood in wounds and tissues present.	No clotting or soft clot.
(6) Vital reaction:	Signs of vital reaction present, i.e., inflammation and repair.	No signs of vital reaction.
(7) Enzyme histochemistry:	Increased activity of adenosine triphosphate, aminopeptidase, acid and alkaline phosphatase.	Diminished or no enzyme activity.

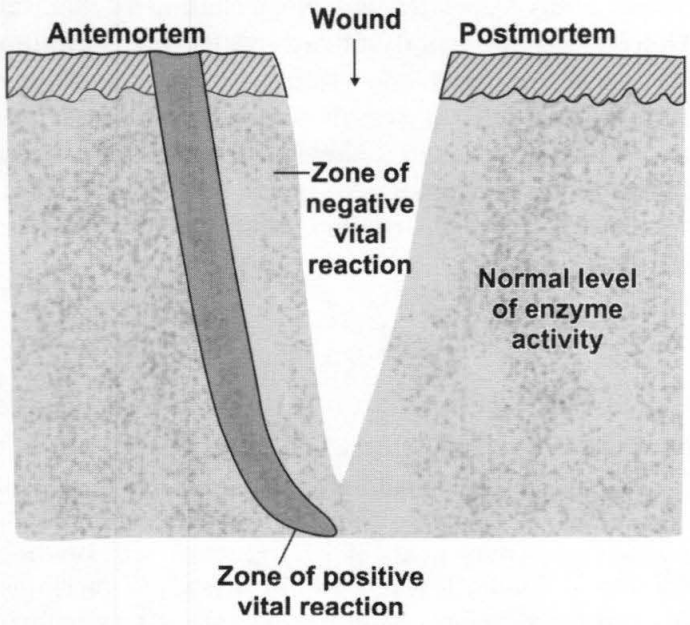


Fig. (10-3). Histochemical wound changes.

within a few hours. Mitotic division is seen in fibroblasts about 15 hours after the injury. Vascularised granulation tissue develops in 72 hours. New collagen fibrils are formed within 4 to 5 days.

(2) Can the time of infliction of the wound be determined?

WOUND HEALING: The morphological sequences of wound healing can be divided into four periods. (1) the phase of traumatic inflammation which lasts from one to three days after wounding, characterised by the appearance of fibrin and dilated capillaries, (2) a destructive phase, from 4 to 6 days, characterised by numerous leucocytes and macrophages, (3) a proliferative phase, from 4 to 14 days, characterised by fibroblasts around capillaries and some metachromasia of tissue ground substance; and (4) a phase of maturation which is gradually reached over many months, in which collagen is found with decrease in the number of fibroblasts, and a progressive increase in tensile strength of the wound. An educated estimate is the best that can be expected.

(3) Which of the several injuries caused death?

The actual cause of death must be found out, and when there is more than one wound, it may be necessary to determine which one of them caused death, since the wounds may not have been made by the same assailant, nor at the same time. This may be done by examining the wounds individually and noting which of them involved injury to some vital organ or large blood vessel or led to secondary results causing death.

(4) How long did the victim live, and could he have carried out voluntary acts after receiving a fatal injury?

Individuals vary in their reaction to injuries. In severe injuries of the brain, sometimes a person survives, although the damage done might have been expected to have caused instant death. A person may remain conscious for several minutes before dying from a severe intracranial injury. Sometimes, a person may die from a blow which produces no naked-eye changes in the brain. Stab wounds of the heart are dangerous, but often rapidly disabling or fatal. Immediate death is likely when the auricles are extensively torn or punctured, but in less severe wounds, powers of voluntary acts and locomotion are retained for some time. Muscular powers are retained in ruptures of liver, spleen or kidneys, unless there is marked immediate blood loss. When the windpipe is divided, the voice is usually lost.

(5) What is the relationship of trauma and natural disease?

There is strong evidence that trauma does not produce neoplasia and that the coexistence of the two processes is purely coincidental. Often, trauma merely calls attention to a preexisting tumour that had not previously been noted. In rare

cases, there is apparent relationship between tumour and some preceding trauma to the part. Since trauma disrupts tissue, it might activate a preexisting tumour to grow and spread more rapidly. Repeated minor trauma is called chronic irritation. With repeated minor trauma and disruption of tissue, the cellular proliferations of regeneration and repair continue. In few cases, cancer develops after many years, e.g., cancer of the skin adjacent to a chronic osteomyelitic sinus that has drained for a number of years. Skin cancer developing in burn scars has been well recognised.

Myocardial infarction can be precipitated by severe physical effort or mental strain in a person with pre-existing coronary heart disease. If the attack occurs within seconds or minutes after unusual effort, the causal connection can be established. Causal connection can be established with certainty only in direct trauma to the heart occurring during work. Attacks occurring few days later may be due to a haemorrhage in an atherosclerotic plaque in the coronary artery which initially narrows the lumen, but later causes occlusion.

CHAPTER

11 Thermal Deaths

COLD

Exposure to cold produces hypothermia which is defined as an oral or axillary temperature of less than 35°C.

LOCAL EFFECTS: (1) Trench foot and immersion foot: They occur due to prolonged exposure to severe cold (5 to 8°C) and dampness, such as is typically seen in soldiers during winter warfare, especially in trenches and in persons exposed to prolonged immersion. The extremities are affected in those conditions. (2) Frostbite occurs due to exposure to extremes of cold (–2.5°C), develops more rapidly and in addition to the extremities, it frequently affects other parts, e.g., nose, ears and the face. In frost-bite, necrosis with blister formation and gangrene occurs.

GENERAL EFFECTS: Ill-effects appear in three stages. (1) The patient feels cold and shivers and the body temperature falls. (2) Shivering stops when the temperature is at or below 32°C. The victim becomes lethargic, drowsy and sleepy, which gradually passes into stupor and coma. The muscles stiffen and mobility is impaired. If he tries to walk, he may feel as if drunk. Respiration, circulation, metabolic and enzymatic processes and oxygenation of all cells are slowed down or blocked. (3) The temperature is lowered to 27°C or even less, which if maintained for 24 hours or longer is fatal. Death results from the failure of the vital centres due to anoxia.

PARADOXICAL OR RECIPROCAL UNDRESSING: This is a very rare and strange condition linked with accidental hypothermia, especially in old persons. The persons take off some or all of their clothing. The cause of the condition is not known, but it may be due to long exposure to severe cold which causes failure of vasoconstriction of arterioles of the skin, which results in a flow of blood from the central part of the body, thus giving an exaggerated sensation of warmth. Because of the great discomfort, the person may undress himself. In such case, there may be suspicion of sexual offence.

THE HIDE-AND-DIE SYNDROME (Terminal burrowing behaviour): In some cases of hypothermic deaths, the disoriented, dying, hypothermia victim, who while attempting to protect himself from the cold may hide himself in corners, cupboards or under piles of furniture or household goods. More commonly, a naked or partly clothed old person is found amid a scene of utter confusion with furniture pulled over and drawers and cupboards emptied out. This may lead to suspicion of homicide and robbery.

NEONATAL COLD INJURY: It results from a failure of the metabolism to prevent a fall in temperature in a body kept in an unsuitable cold environment. The symptoms appear usually in the first week of life. The rectal temperature is usually below 32°C. The outstanding feature is swelling and widespread oedema of the extremities, particularly the hands and the feet, and the eyelids. The face, hands and feet are red, which is a striking and constant finding. Localised hardening of the skin and subcutaneous tissue overlying oedematous parts starts in the distal parts of the legs and spreads to involve the trunk. Pulsation of peripheral arteries is absent. Respirations are shallow, slow and may be irregular. Infants lie motionless, though conscious. The haemorrhagic tendency is manifested by gastrointestinal bleeding, petechial haemorrhages and oozing of blood from scratches and injection sites.

MEDICOLEGAL IMPORTANCE: It is a method of killing illegitimate and unwanted infants.

HEAT

Three clinical conditions may result from exposure to high environmental temperature: (1) heat cramps, (2) heat prostration, and (3) heat hyperpyrexia.

(1) HEAT CRAMPS: (miner's cramps, stoker's cramps, or fireman's cramps): They are caused by a rapid dehydration of body through the loss of water and salt in the sweat. It is seen in workers in high temperature when sweating has been profuse. The onset is usually sudden. Severe and painful paroxysmal cramps affecting the muscles of the arms, legs and abdomen occur.

(2) HEAT PROSTRATION (heat exhaustion or heat syncope, or heat collapse): Heat prostration is a condition of collapse without increase in body temperature, which follows exposure to excessive heat. It is precipitated by

muscular work and unsuitable clothing. There is extreme exhaustion and peripheral vascular collapse. The face is pale, the skin cold, the temperature subnormal. The pupils are dilated, the pulse small and thready and the respiration sighing. The patient usually recovers if placed at rest, but death may take place from heart failure.

(3) Heat Hyperpyrexia or Heat Stroke: Heat stroke is characterised by **rectal temperature greater than 41°C., and neurological disturbances**, such as psychosis, convulsions, delirium, stupor, coma. The term **"thermic fever or sunstroke"** is used when there has been direct exposure to the sun.

Precipitating factors: High temperature, increased humidity, minor infections, muscular activity, and lack of acclimatisation are the principal factors in the initiation. Where there is 100% humidity, a temperature of 32°C in the environment may lead to heat stroke. Failure of cutaneous blood flow and sweating, the factors which control body temperature, leads to a breakdown of the heat regulating centre of the hypothalamus.

Heat stroke usually occurs: (1) in young person's exposed to high temperature while undergoing severe exertion, and (2) in old persons usually over 60 years during heat waves.

Types: (1) **Exertional heat stroke** most commonly occurs in athletes, military personnel working hard in a hot environment. A persistent sweating as a result of increased catecholamine production can be seen in about 50% of individuals. (2) In the **classic form** sweating is usually absent.

CLINICAL FEATURES: The onset is usually sudden, with sudden collapse and loss of consciousness. The skin is dry, hot and flushed with complete absence of sweating. The pupils are contracted. The pulse is rapid and later becomes irregular. The breathing is rapid, deep and of Kussmaul type. Blood pressure is low. Convulsions occur and the patient becomes delirious or comatose. The fatal period is five minutes to three days.

Postmortem Appearances: They are not specific. The temperature remains high after death. The brain is congested and oedematous and petechial haemorrhages are seen in the white matter. Trachea and bronchi contain frothy haemorrhagic fluid. The lungs show oedema, congestion and haemorrhages. Internal organs are congested. Petechial and larger haemorrhages are seen in most organs.

BURNS

A burn is an injury which is caused by application of heat or chemical substances to the external or internal surfaces of the body, which causes destruction of tissues. The minimum temperature for producing a burn is about 44°C for an exposure of about five to six hours. **At 65°C, two seconds are sufficient to produce burns.**

Varieties of Burns: (1) A highly **heated solid body** or a molten metal, when applied to the body for a very short time produces only a blister and reddening, corresponding in size and shape to the material used. It will cause destruction, or even charring of the parts, when kept in contact for some time. The epidermis may be found blackened, dry, and wrinkled. The hair may be singed or distorted. Hair singed by the flame becomes curled, twisted, blackish, breaks off or is totally destroyed. (2) Burns produced by **flame** may or may not produce vesication, but singeing of the hair and blackening of the skin are always present. Roasted patches of skin or deeper parts may be seen. (3) Burns caused by **kerosene oil**, petrol, etc. are usually severe and produce sooty blackening of the parts and have a characteristic odour. (4) Burns caused by **explosions** in coal mines or of gunpowder are usually very extensive and produce blackening and tattooing due to driving of the particles of the unexploded powder into the skin. (5) Burns due to **X-rays and radium** vary from redness of the skin to dermatitis, with shedding of hair and epidermis and pigmentation of the surrounding skin. Severe exposure may produce burns with erythema, blistering or dermatitis, or ulceration with delayed healing and ill-formed scars. Infrared rays may cause necrosis of the skin. (6) Burns caused by **ultraviolent rays** (the sun or mercury vapour lamp) produce erythema or acute eczematous dermatitis. (7) **Radiant-heat burns** are caused by heatwave, a type of electromagnetic wave. (8) **Microwave burns** are well-demarcated, full thickness burns. (9) Burns from **corrosive substances** show ulcerated patches and are usually free from blisters; hair is not singed and the red line of demarcation is absent. They show distinct colouration, and are usually uniform in character. Strong acids produce dark leathery burns upon the skin. Strong alkalis cause the skin to slough and leave moist, slimy, greyish areas. (10) **Electrical burns.**

Degree of Burns: Dupuytren divided burns into six degrees but they are merged into three degrees by Wilson.

(1) Epidermal: The affected part is red. **Usually a blister (vesicle or bulla) is formed which is covered by white, avascular epidermis and bordered by red, hyperaemic skin usually 5 to 20 mm, across.** The base is bright red when burst. Singeing of the hair is present. The blister contains gas and protein-containing fluid. When epidermis is lost, the dermis becomes reddened, inflamed and exudes plasma and tissue fluid. These burns are very painful. Repair is complete without scar formation.

(2) Dermo-epidermal: Whole thickness of skin is destroyed. These burns appear as wrinkled, depressed areas of coagulated tissue, bordered by reddish, blistered skin. The lesions may be brown or black, due to charring and eschar formation. The necrotic tissue separates usually within a week and leaves an ulcer, which heals with scar formation. Pain and shock are greater than in first degree burns.

(3) Deep: In this, there is a **gross destruction not only of the skin and subcutaneous tissues, but also of muscles and even bone.** Nerve endings are also destroyed, and as such, the burns are relatively painless. **The appearances are similar to those of the second degree, but in a more severe form. The burnt part is completely charred (Plate 15-86 and 15-87).**

Clinical classification: (1) Superficial, when less than full thickness of skin is involved. (2) Deep.

Effects: The effects depend on: **(1) The degree of heat:** The effects are severe if the heat applied is very great. The body of an adult does not burn completely in a burnt house as the temperature usually does not exceed 650°C. For purpose of cremation, a human body has to be incinerated for one-and-half hours at 1000°C. The ashes weigh two to three kg. and contain bone fragments which can be identified as human. **(2) The duration of exposure:** The symptoms are more severe if the heat is applied for long time. **(3) The extent of the surface:** The estimation of the surface area of the body involved is usually worked out by the **"rule of nines"**, 9% for the head (head 7%, neck 2%) and each upper limb, (arm 4%, forearm 3%, hand 2%), 9% for the front of each lower limb 9% for the back of each lower limb, (thigh 10%, leg 5%, foot 3% for each lower limb), 9% for the front of chest, 9% for the back of the chest, 9% for the front of the abdomen, and 9% for the back of the abdomen, 99% of the body. The remaining one percent is for the external genitalia. **(Table 11-1 and Fig. 11-1). Palmar surface of patient's hand is about one percent of body surface area.** Involvement of 50% of the body surface will prove fatal even when the burns are only of the first degree. **(4) The site:** Burns of the head and neck, trunk or the anterior abdominal wall are more dangerous. **(5) Age:** Children are more susceptible, old people less. **(6) Sex:** Women are more susceptible.

Causes of Death: (1) Primary (neurogenic) shock. (2) More than half of the deaths occur within the first 48 hours, usually from secondary shock due to fluid loss from burned surface. Circulatory collapse may occur with 15% of burns of total body surface area. (3) Toxaemia, due to absorption of various metabolites from the burnt tissue, persists up to three to four days. (4) Sepsis is the most important factor in deaths occurring 4 to 5 days or longer after burning. (5) Biochemical disturbances, e.g., hypokalaemia. (6) Acute renal failure due to lower nephron nephrosis occurs on the third or fourth day. (7) Oedema of glottis and pulmonary oedema due to inhalation of smoke containing CO and CO_2 if the person dies in a burnt house. (8) Accident occurring in an attempt to escape from a burning house or by injuries due to falling masonry, timber or other structure on the body. (9) Pulmonary embolism from thrombosis of veins of the leg due to tissue damage and immobility. (10) Death may occur years after recovery from malignant transformation of a burn scar **(Marjolin's ulcer)**.

Table (11-1). Estimation of percentage of body surface area burned (Lund and Browder)

Area of Body	1 to 4 yrs	5 to 9 yrs	10 to 14 yrs	Adult
Head and neck	**19**	**15**	**13**	**09**
Front of trunk	**16**	**16**	**16**	**18**
Back of trunk	**16**	**16**	**16**	**18**
Upper limb	**19**	**19**	**19**	**18**
Lower limb	**30**	**34**	**34**	**36**
Genitalia		**0**		**1**

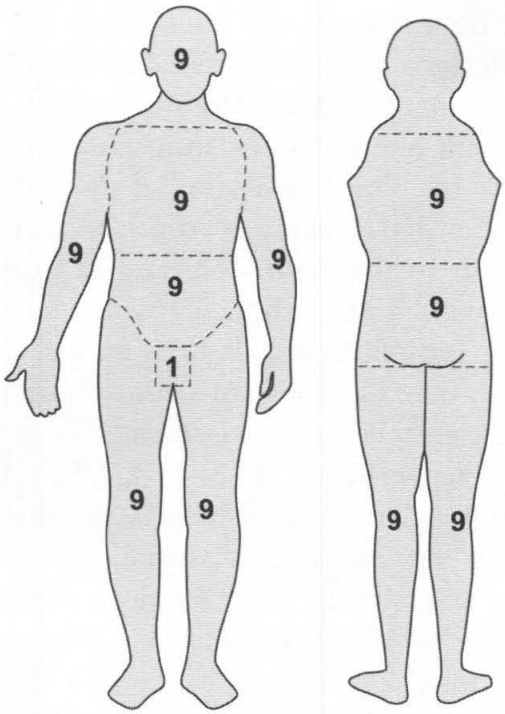


Fig. (11-1). Estimation of the extent of surface burns.

Postmortem Appearances: External Clothes: Cotton fabrics ignite easier and burn faster than other types of fabrics. Nylon, polyester, and wool produce less severe burns. **Close fitting garments tend to be safer than long, loose garments.** The clothes should be removed and examined for the presence of smell of kerosene, petrol, etc. They should be put into airtight bottles or in plastic bags and sent for chemical analysis.

Heat rigor may be observed in the muscles. It is **difficult to determine the time of death as body temperature, postmortem hypostasis and rigor mortis cannot be assessed.** The face is swollen and distorted. The tongue protrudes due to the contraction of the tissues of the neck and face. In the hands, the skin detaches as glove, including the fingernails. **Flame burns** usually have a patchy distribution and vary in size and shape. **"Flash burns"** are a variant of flame burns. **They occur due to sudden ignition or explosion of gases or fine particulate material,** e.g. explosions, or ignition of highly inflammable liquids. **All exposed surfaces are burned uniformly.** If clothing is ignited, a combination of flash and flame burns occur. The burnt areas will be found reddened and blistered or charred. Hair is singed. The degree of burning in each area should be assessed. **Blisters** may be present either in the main burn or as islands beyond the periphery. **The blisters of a second degree burns cannot be distinguished from blisters seen in CO poisoning, deep coma, exposure to gasoline and putrefaction.** When these various types of blisters burst, they leave a pale, moist, raw surface which becomes yellow, tan and finally dark-brown and leathery as it dries. In severe burns, muscles are shrivelled and desiccated even carbonised on the surface. Beneath that there is a zone of brownish pink cooked meat and under that normal red muscle.

Pugilistic attitude (boxing, fencing or defence attitude): The posture of a body which has been exposed to great heat is often characteristic. **The legs are flexed at the hips and knees, the arms are flexed at elbows and held out in front of the body, head slightly extended, all fingers are hooked like claws.** Contraction of paraspinal muscles often causes a marked opisthotonus, in an attitude commonly adopted by boxers **(Plate 15-88)**. This stiffening is due to the coagulation of proteins of the muscles, and dehydration which causes contraction. The flexor muscles being bulkier than extensors contract more due to which joints of all limbs are flexed. It occurs whether the person was alive or dead at the time of burning.

Heat Ruptures: If the heat applied is very great, skin contracts and heat ruptures occur, either before or after death. They are produced by splitting of the soft parts. These splits may be anywhere, but are usually seen over extensor surfaces, joints and on the head. **These ruptures or splits of skin may be several centimetres in length, and superficially they may resemble lacerations or even incised wounds. They can be differentiated by:** (1) Absence of bleeding in the wound and surrounding tissues, since heat coagulates the blood in the vessels. (2) Intact vessels and nerves are seen in the floor. (3) Irregular margins. (4) Bruising or other signs of vital reaction are not present in the margins.

Sometimes, **the charred skin cracks easily** when an attempt is made to remove the body from a house destroyed by fire **(Plate 16-96)**. These tears are commonly seen around joints, especially the elbows, shoulders and knees. Portions of the body where clothing is tight, e.g., under the belt, shoes, brassier or buttoned collar are often comparatively unaffected.

Partial burning of the abdominal wall associated with gas expansion within the intestines may produce rupture of the abdominal wall in the charred burnt victim. The intestines may protrude through this defect. Areas where the burning is severe may be completely destroyed. If the flame is unchecked, the body will be reduced to a shapeless, coal-like mass and finally to heap of grey and yellow ashes.

Establishment of Identity: In a charred body stature may be less by several centimetres and weight loss may be up to 60%. The features are changed due to contractions of the skin. Moles, scars and tattoo marks are usually destroyed. Complete X-rays of body of the victim are useful to locate possible old fractures, bony abnormalities or foreign bodies. In a badly charred or incinerated body, the sex can be determined by finding the uterus or prostate which resists fire to a marked degree and by pelvic bones, and age by teeth and observing centres of ossification in the bones and the condition of epiphyses. If the whole body is destroyed, personal effects, such as keys, watch strap, buttons, belt-buckle, cuff-links, etc. may help in identity. DNA extracted from blood from cardiac chambers or from any human remains is analysed using PCR technique and compared with the alleged biological parents.

Internal: Heat haematoma: occurs when the head has been exposed to intense heat, sufficient to cause charring of the skull. It has the appearance of extradural haemorrhage, but is not accompanied by any signs of injury by blunt force. **It consists of a soft, friable clot of light-chocolate colour, and may be pink if the blood contains CO. The clot has a honeycomb appearance due to bubbles of steam produced by heat. The thickness of the clot varies from one to fifteen mm. and the volume up to 120 ml. The distribution of the clot follows closely the distribution of the charring of the outer table of the skull.** The dura underneath the haematoma is greatly shrunken which compresses the brain. The dura may split under tension and brain tissue may ooze out and form a mass of frothy paste. The parietotemporal region is the most common site of such haemorrhage (**Table 11-2, Fig. 11-2 and Plate 16-92**). The mechanism of its development is obscure. It may be due to expansion of blood in the skull diploe and the intracranial venous sinuses which rupture, or shrinkage of meninges and brain, which may aspirate blood from the venous sinuses. The carboxyhaemoglobin level in heat haematoma will be the same, as in peripheral blood.

Thermal fractures of the skull: (1) The fracture results from the rapid increase in intracranial steam pressure and the fragments are displaced upwards. (2)

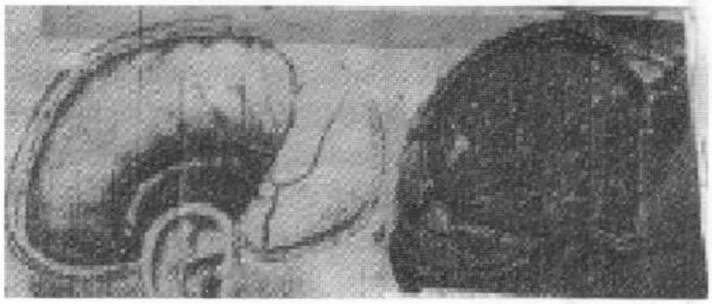


Fig. (11-2). Extradural haemorrhage and heat haematoma.

Table (11-2). Difference between extradural haematoma due to burns and due to blunt force

Trait	Extradural haematoma due to burns	Extradural haematoma due to blunt force
(1) Cause:	Charring of the skull due to intense heat.	Blunt force to the head.
(2) Situation:	Anywhere.	Usually adjacent to sylvian fissure.
(3) Distribution:	Usually bilateral. Diffuse.	Usually unilateral. Localised.
(4) Characters:	Thin, granular, soft, friable clot of light chocolate colour, honeycomb appearance; evenly distributed or sickle-shaped.	Discoid shape: localised, more rubbery consistency, reddish-purple.
(5) Skull:	May be fractured.	Associated fracture in temporal area.
(6) CNS:	No injury.	Frequently injured.
(7) Carboxyhaemoglobin:	Present.	Absent.

Fig. (11-3). Heat fractures of the skull due to burns.

The fracture occurs due to rapid drying of the bone with contraction, and only involves the outer table of the skull. In this type, there is no displacement and the lines of fracture are frequently stellate. Skull fractures are usually seen on either side of the skull above the temples. They consist of several lines which radiate from a common centre. Heat fracture may cross a suture line **(Fig. 11-3)**. Peculiar, characteristically curved fractures are often seen in bones of extremities exposed to very high temperatures. Teeth crack when exposed to heat. Enamel crowns are completely lost, while roots remain intact but opaque.

CO levels: In death due to burns, the CO levels in the blood will be more than 10%, and may reach 70 to 80%. The blood is cherry-red which may change to brownish due to heat. CO may be absent in blood due to rapid death, convection air currents, low production of CO, flash fire (as in the fire of a chemical plant), inhalation of superheated air resulting in death by suffocation, in warfare, or in an explosion when death is instantaneous.

Death from suffocation: If death has occurred from suffocation, aspirated blackish coal particles are seen in the nose, mouth, larynx, trachea, bronchi, oesophagus and stomach, and blood is cherry-red. Such particles are embedded in frothy mucus which covers the congested mucosa **(Plate 16-95)**. **Presence of carbon particles especially in the terminal bronchioles and an elevated CO saturation together are absolute proof that the victim was alive when the fire occurred.** In the absence of CO in the blood and soot in the airways, death may result possibly due to poisoning with CO_2 and/or O_2 deficiency. Sometimes inhalation of smoke produces vomiting which may be inhaled and found in the smaller bronchi.

Viscera: Haemoconcentration is present and frequently there is some tissue oedema and excess of fluid in the serous cavities. **Even in cases of severe external charring, the internal organs are usually well preserved, as the tissues of the body are poor heat conductors. Sometimes, brain, liver, lung, etc. may be cooked, i.e., hardened and discoloured.** The brain is usually shrunken, firm and yellow to light-brown due to cooking. The dura mater is leathery. The pleurae are congested and inflamed. The lungs are usually congested, may be shrunken and rarely anaemic. The vessels of the lungs may contain a small amount of fat due to a physico-chemical alteration of fat already present in the blood. The lungs are heavy, airless and consolidated. They may be hardened and discoloured. Signs of infection and inflammation may be present in delayed death. Visceral congestion is marked in many cases. Petechial haemorrhages are usually found in the pleurae, pericardium and endocardium. The heart is usually filled with clotted blood. **There may be inflammation and ulceration of Peyer's patches and solitary glands in the intestines. Occasionally, ulcers are produced in the duodenum (Curling's ulcers) about the tenth day in extensive burns of the body.** Curling's ulcers are usually sharply punched-out mucosal defects, which may be superficial or deep. Petechiae of stomach and duodenum, often with erosions, occasionally acute ulcers are a more common finding. The large bowel may also be involved. The spleen is enlarged and softened. The liver may show cloudy swelling. Jaundice may occur. The kidneys may show cloudy swelling, capillary thrombosis and infarction. The adrenals may be enlarged and congested. **When more than 30% of the skin surface is burnt, haemoglobinuria occurs.** Depending on the materials burning in the fire, various levels of **cyanide are found in the blood; but the levels are usually less than 0.3 mg%.**

Age of Burns: The ageing of the burns is very inaccurate and depends upon the agent, the extent and their depth. Redness appears immediately and vesication in about an hour. The exudate begins to dry in 12 to 24 hours and forms a dry, brown crust within 2 to 3 days.

The red inflammatory zone disappears in 36 to 72 hours, and pus may form under sloughs. Superficial sloughs fall off in 4 to 6 days, and deeper sloughs within 2 weeks. After this, granulation tissue covers the surface and a scar is formed after several weeks (Plate 16-91).

Antemortem and Postmortem Burns: In antemortem burns, a zone of hyperaemia (line of redness), which varies in width but is usually about a centimetre is present at the edge of the burnt area, except in cases of immediate death. It involves whole thickness of true skin. It is permanent and persists after death (Plate 15-89). If the person is alive, the skin beyond this zone shows erythema, which tends to disappear after death. **If the whole body is burnt, line of redness will be absent.** The antemortem blister appears as a raised dome and contains gas or fluid. The base and periphery show reddening with swollen papillae. In antemortem burns the skin adjacent to burnt area shows an increased reaction for SH groups in all layers, and increase in enzyme reaction. Acid mucopolysaccharides are present in the superficial zone of burnt area. Postmortem burns show dried scorching of the skin surface, like burnt paper. The base of the postmortem blister is dry, hard and yellow (Plate 15-90). Burns produced shortly before or after death cannot be distinguished either by naked eye or by microscopic examination (Table 11-3).

Teeth: When a living person is burnt, the teeth become loose in their sockets, whereas in a dead person the teeth are firmly attached.

CIRCUMSTANCES OF DEATH

ACCIDENT: Large numbers of deaths are accidental. Infants, children, epileptics, intoxicated or drugged persons or helpless from other causes may fall into a fire. When an intoxicated person goes to bed smoking, and drops a lighted cigarette, he may die due to burns. Lamps or stoves may explode and set fire to the clothes. Clothes of women may catch fire accidentally while cooking. Multiple deaths from burns may result from plane crashes or automobile accidents. In industry, burns may be caused by explosions from inflammable liquids and by flashes from furnaces.

SUICIDE: Occasionally, women commit suicide by pouring kerosene on their clothes before setting fire to themselves due to domestic worries, cruelty by the husband or other family members, disappointment in love or acute or chronic disease. Sometimes, suicidal burning is a mode of public protest.

HOMICIDE: Murder by burning is rare. An inflammable fluid, such as kerosene, petrol, etc. may be poured on a person and then burnt. Sometimes, fire, hot metals, boiling water and corrosive substances are used with criminal intent. An angry drunken man may push or throw his wife or child on the fire and sometimes lighted lamps may be used as missiles. Burns may be inflicted on the pudenda of women as a punishment for adultery. Attempts may be made to burn a body after homicide with the object of concealing the crime. In such cases, the body should be examined for marks of violence, e.g., stab wounds, bullets, strangulation, etc.

Self-inflicted Burns: Burns are sometimes self-inflicted in order to support a false charge.

Spontaneous Human Combustion: (preternatural combustion): A human body may sometimes burn away almost completely, with minimal damage to the surrounding area. This invariably occurs near a hearth, or open fire-grate or chimney. The burning may be confined to the body, its clothing and a narrow zone of floor or carpet. Often the victims are alcoholic and confused, instable and lack judgement. It appears that when a person collapses and falls and part of the body comes in contact with a source of heat, clothing catches fire and burns the limited amount of oxygen available in the room, after which that part is ignited,

Table (11-3). Difference between antemortem and postmortem burns

Trait	Antemortem burns	Postmortem burns
(1) Line of redness:	Present.	Absent.
(2) Blisters:	Contain serous fluid with proteins and chlorides. Base is inflamed.	Contain air and thin fluid only. Base is dry, hard and yellow.
(3) Vital reaction:	Marked cellular exudation and reactive changes in the tissue cells present.	Absent.
(4) Enzymes:	Peripheral zone of burn shows increase in enzyme reaction.	Peripheral zone does not show increase in enzyme reaction.

and adjacent body fat melts and soaks into clothes. The clothing acts as a wick, melts the next zone of adjacent fat and the process is repeated along the length of the body, till the joints of extremities are reached and the burning stops because of less fat and also due to less layers of clothes. Often, all soft tissues are reduced to ash and most of bones are calcined. The only part of body left undamaged is likely to be one or both of lower legs. Usually CO is not found in the blood. The room in which the body is found is frequently very hot and the contents, walls, furniture, etc are heavily soiled by soot, but there is no fire damage to any of the objects in the room.

SCALDS

A scald is an injury which results from the application of liquid above 60°C. or from steam.

Degrees of Scalds: (1) Erythema by vasoparalysis. (2) Blister formation due to increased permeability of the capillaries. (3) Necrosis of the dermis.

Features: Redness appears at once, which resembles a first degree dry burn, but the shape of scald tends to be different. The severity of a scald depends on duration of contact with the skin and temperature of fluid, and blistering will take place within a few minutes.

Blisters have a hyperaemic zone around them. There is a reddening and swelling of the papillae in the floor of the blister. The blister fluid contains white and red cells. The intensely red base of a severe scald is at first covered with wrinkled, macerated epidermis. If blistered skin is removed, it will leave a pink raw surface and later the exposed dermis becomes brownish, hard and dry. Scalds show soddening and bleaching, but do not singe the hair, and do not blacken or char the skin. Superheated steam soddens the skin which becomes dirty-white colour. **Scalding can occur through clothing.** The injury is limited to the area of contact and is more severe at the point of the initial contact. Scalded areas are usually large, but may be small if caused by splashing. **Streaks of liquid run downwards from the main area causing lines of blisters.** Sticky liquids, such as syrup, oils and tar cause more severe scalds than hot water. Scars of scalds are much thinner than those of deep burns and cause less contraction and disfigurement. A postmortem blister does not show hyperaemia in the surrounding area and the floor is not red (Table 11-4).

Toxic epidermal necrolysis due to diseases or drugs cause blistering and separation of superficial

Table (11-4). Difference between burns from dry heat, moist heat and chemicals

	Trait	Dry heat	Moist heat	Chemicals
(1)	Cause:	Flame, heated solid body, or X-rays.	Steam or liquid above 60°C.	Corrosive chemicals.
(2)	Site:	At and above the site of contact.	At and below the site of contact.	At and below the site of contact.
(3)	Splashing:	Absent.	Present.	Present.
(4)	Skin:	Dry and wrinkled; may be charred.	Sodden and bleached.	May be destroyed.
(5)	Red line:	Present.	Present.	Absent.
(6)	Vesicles:	At the circumference of burnt area.	Over the burnt area.	Very rare.
(7)	Colour:	Black.	Bleached.	Distinctive.
(8)	Charring:	Present.	Absent.	May be present.
(9)	Singeing:	Present.	Absent.	Absent.
(10)	Ulceration:	Absent.	Absent.	Present.
(11)	Scar:	Thick and contracted.	Thin and less contracted.	Thick and contracted.
(12)	Clothes:	Burnt.	Wet; not burnt.	May be burnt; show characteristic stains.

layers of skin with a raw weeping surface has a close similar appearance to a scald.

OCCURRENCE: Scalds are usually accidental due to bursting of hot water bottles, bursting of boilers, splashing of fluid from cooking utensils or immersion in liquid over 60°C. Suicide by scalding is rare. Boiling water may be thrown with intent to injure. Murder by scalding is rare. Steam burns are rare.

ELECTRICAL INJURIES

Electrocution is death caused by passage of electric current.

Effects: The injuries caused by contact with electrical conductors depend upon: (1) **The kind of current:** Alternating current is four to five times as dangerous as an equal voltage of direct current. (2) **The amount of current:** Electrocution is rare at less than 100 volts, and most deaths occur at more than 200 volts. High voltages may cause the victim to be thrown clear, while lower tensions around 240, cause muscle contraction, due to which the victim holds the source of current. (3) **The path of the current:** Death is more likely to occur if the brain stem or heart is in the direct path of the current. (4) **Duration of the current flow:** The severity is directly proportional to the duration of current flow.

Electrocution: For an electric shock to occur, there must be contact by the body with both a positive and negative pole, or alternatively, the "earth". The earth may be any object not insulated from the ground. When earthing of body is poor, as with dry and rubber-shoes, carpets, wooden floors and upstair premises, fatal electrocution is uncommon.

Local Effects: (1) The electric mark (Joule burn): It is specific and diagnostic of contact with electricity and is found at the point of entry of the current. It is endogenous thermal burn due to the heat generated in the body from electricity. **These marks are round or oval, shallow craters, one to three cm. in diameter, and have a ridge of skin of about one to three mm. high, around part or the whole of their circumference. The crater floor is lined by pale flattened skin.** In some marks the skin may break within or near the margin of the crater, resembling that of a broken blister. When contact is more prolonged, the skin in the mark becomes brown, and with further contact, there may be charring. If the conductor contains copper, the electrical mark can

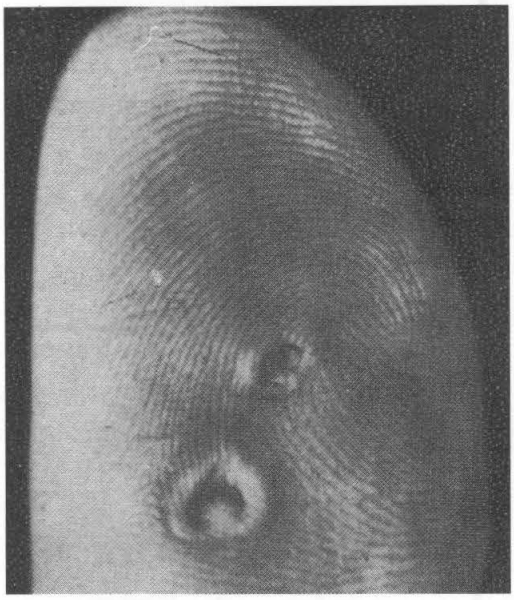


Fig. (11-4). Electric mark on the fingers.

have a bright green colour **(Fig. 11-4).** Where the tip of the wire or rod is at right angle to the skin, the mark may be present as a circular hole penetrating skin, muscle and even bone, so as to simulate a bullet hole. Rarely, the mark may be present as a circular hole, penetrating skin, muscle, and even bone, so as to simulate a bullet wound. These electric marks are produced by the conversion of electricity into heat within the tissues. They are commonly found on exposed parts of the body, especially on the palmar aspect of the hands.

(2) **Flash or Spark Burns:** They are due to **contact with voltage in excess of thousand volts. The intense heat which may result from flashover produces burns, which resemble thermal burns.** The burns may be as small as pinpoint, or deeply seated and contracted, if contact is prolonged or very high voltage is applied. When bone is involved, periosteum may be destroyed or fracture may occur. Sometimes, multiple lesions are found in the region of flexures of limb where the current has passed across the joints, instead of passing around it. High tension electrical currents may produce multiple discrete lesions due to arcing from the conductor to the body without direct contact. **Multiple burnt or punched-out lesions are produced due to the arc dancing over the body surface over large areas, which present crocodile flash burns (Plate 17-97).** High amperage has an explosive blast effect and may produce injuries resembling bullet, stab or incised wounds.

Burns may be absent: (1) If the area of contact is relatively large, e.g., when a hot wire is grasped with a wet hand, or when a person is electrocuted in bath tub. (2) When the area of contact is small in low-voltage electrocutions. (3) Brief contact with a live wire may not produce burns. In this case, the person may collapse from ventricular fibrillation and fall away from the wire.

(3) **Electric Burns or Splits:** When the electrical conductor is a wire, a linear burn may occur. When the current passes from a metal conductor into the body, metallic ions are embedded in the skin and even in subcutaneous tissues. The splits are dry, hard, firm, charred, insensitive with ragged edges and their form is round, oval, linear, or of irregular shape. The depth of the lesion is much greater than appears on the surface. Wrinkling of the skin may be found, and occasionally localised oedema of a limb. Aseptic necrosis develops, which often extends beyond the burns in area and depth and may lead to sloughing.

Exit Marks: These are variable in appearance, but they have some of the features of entrance marks. There may be more damage of tissues, and they are often seen as splits in the skin at points where the skin has been raised into ridges by the passage of current; splitting of these ridges may be continuous or interrupted.

Postmortem Appearances: External: The **examination of the scene may be much more important than the postmortem of the body.** The **clothing** including shoes, gloves and head gear should be examined for burns. Rigor mortis appears early, and postmortem lividity is well developed. The **face** is pale, the eyes are congested and pupils dilated. Usually there are external marks of electric burning and contusion or laceration at the point of entrance and exit of the body. In some cases, the lesions may extend through subcutaneous tissues and involve muscles and bone. A number of greyish-white circular spots, which are firm to the touch and free from zone of inflammation, may be found at the site of entrance and exit. Extensive ecchymosis may be found on the skin of the trunk. **Current marks may be hidden inside the oral cavity**, from putting live wire into the mouth or from drinking at a water fountain in contact with electric current. They may be found in the **urethra** due to urination on a high tension wire. **Any metallic object on the body will produce corresponding burns on the skin because it becomes heated by the passage of the current.** Severe convulsions caused by electrical discharge may cause fractures of the spine or limbs. In some cases, the entrance and exit marks cannot be determined grossly.

Internal: The appearances are usually those of **asphyxia**. The lungs are congested and oedematous and the brain, meninges and parenchymatous organs are congested. Petechial haemorrhages may be found along the line of the passage of the current, under the endocardium, pericardium, pleura, brain and the spinal cord. There may be necrosis of the intima or of the complete wall of the blood vessels. Vascular thromboses may be seen in the vicinity of electrical burns. **High amperage has an explosive effect and may produce injuries resembling bullet, stab or cut wounds.** Small balls of molten metal, derived from the metal of the contacting electrode, so-called **current pearls**, may be carried deep into the tissues. Heat generated by the current may melt the calcium phosphate, which is seen radiologically as typical round density foci (**"bone pearls" or "wax drippings"**). There may be bone necrosis, and zigzag microfractures. A foetus may survive the electrocuted mother, or a surviving mother may abort after electric injury. **Occasionally, no lesions can be found either externally or internally.**

Cause of Death: (1) Paralysis of medullary (respiratory) centres. (2) Ventricular fibrillation or cardiac arrest without fibrillation. Death need not be instantaneous. Individuals may be able to walk some distance and talk before the onset of collapse and death. **Death may occur after a few days from infection, or from haemorrhage due to damage to blood vessels.**

Medicolegal Aspects: Death by electric currents are **usually accidental** from defective electric appliances, or negligence in the use of equipment. In industry, deaths may result from contact with live overhead cables or from handling of charged lamps, tools or switch gears. Rarely, death may occur **during convulsive therapy to mental patients (iatrogenic)**. Cases of suicide and even homicide have occurred.

JUDICIAL ELECTROCUTION: Death penalty is carried out in the electric chair, in some States in the USA. The condemned person is strapped to a wooden

chair and one cap-like electrode is put on the shaven scalp, which is moistened with a conducting paste, and the other on the right lower leg, and a **current of 2,000 volts and seven amperes is passed for one minute** through the body. After tetanic spasm and loss of consciousness, **the same current is passed through the body a second time for one minute.** The brain is heated up to 60°C and vacuolation occurs around the vessels.

LIGHTNING STROKE

A flash of lightning (atmospheric electricity) is due to an electrical discharge from a cloud to the earth. The electric current is direct with a potential of about twenty thousand amperes and one thousand to thousand million volts or more. Lightning differs from ordinary electric current only in degree. It is attracted by the highest points. It passes normally along the outside of a conductor, and as such persons in buildings are relatively safe from electrocution. Dry skin and dry clothes are bad conductors, whereas wet skin and wet clothes are good conductors.

Postmortem Appearances: The **clothes** are usually burnt or torn at the point of entrance and exit. In some cases, the clothes may be stripped off the body and thrown to some distance. In exceptional cases, clothing is not damaged even though the person is killed by lightning. Conversely, clothing may be burnt without any injury to the person. Rigor mortis may appear soon and pass off quickly. The expanded, displaced air causes disruptive or **blast-like lesions,** e.g., contusions, lacerations, fractures, ruptures of organs, wounds of almost any variety, burns, etc.

The burns may be: (1) Linear: These vary from 3 to 30 cm. or more in length, and 0.3 to 2.5 cm. in width. They are often found in the moist creases and folds of the skin.

(2) ARBORESCENT OR FILIGREE BURNS: (Lichtenberg's flowers): **They are superficial, thin, irregular and tortuous markings on the skin. These markings have a general pattern resembling the branches of a tree. This fern-like pattern of erythema in the skin is usually found over the shoulders or the flanks.** It is not certain whether it is instantaneous or it develops within few minutes.

Cause: (1) It may be caused due to the slight staining of the tissues by haemoglobin from lysed red blood cells along the path of the electric current, or rupture of smaller blood vessels at several places giving rise to ecchymoses. (2) They may be superficial burns producing mere erythema of skin which indicate the path taken by the current. (3) They are also said to be due to minute depositions of copper in the dermis or from boiling of the intercellular fluid following the fascial planes. They indicate the paths taken by the discharge and disappear in one to two days if the person survives. These marks are seen rarely.

(3) Surface Burns: They are true burns and occur beneath metallic objects worn or carried by the person, which are fused by the flash.

Internal findings are those of asphyxia.

Cause of Death: Involvement of the central nervous system with paralysis of the heart or of the respiratory centre causes death.

Medicolegal Importance: Less than half of the persons struck by lightning are killed. Death is always due to accident. **Sometimes, the appearances left on the human body closely resemble those produced by criminal violence.** Thus, a person may be found dead in an open field or on the highway, and body may show contusions, lacerations and fractures. In such cases, the diagnosis should be based on the history of a thunderstorm in the locality, evidence of effects of lightning in the vicinity of the body, and fusion or magnetisation of metallic substances.

CHAPTER

12 Starvation

Causes: Starvation may occur from the actual stoppage of food or from the administration of unsuitable food.

Types: (1) **Acute starvation** results from sudden and complete stoppage of food. (2) **Chronic starvation** results from gradual deficient supply of food. Starvation deaths may be due to (1) famine, (2) being trapped in pits, mines or landslides, (3) neglect on the part of the parents or guardians, (4) willful withholding of food, and (5) willful refusal to take food.

Symptoms: In acute starvation, there is a feeling of hunger for the first 30 to 48 hours which is followed by pain in the epigastrium, which is relieved by pressure. After 4 to 5 days of starvation, general emaciation and absorption of the subcutaneous fat begins to occur. The eyes are sunken and glistening: the pupils are dilated; the cheeks sink and the bony prominences become prominent. The lips are dry and cracked, and the tongue coated and dirty, and thirst is intolerable. The saliva is thick and scanty. The voice is weak and whispering. The skin is dry, rough, thin, inelastic, wrinkled and pigmented. Emaciation may be extreme, the abdomen concave and the limbs become thin and flaccid with loss of muscular power. Muscular weakness is progressive and may be intense. Cardiovascular changes are those of progressive insufficiency. The pulse is slow at rest, but on exertion paroxysmal tachycardia supervenes. The temperature is subnormal. Constipation is usual, but towards death diarrhoea and dysentery are common. The urine is scanty, turbid, and highly concentrated and shows evidence of acidosis. The loss of weight is most marked and constant. In the last stage, the body is reduced to an extreme state of emaciation and before death has an offensive odour. **Death usually occurs when 40% of the original weight is reached.** The intellect remains clear till death, though in some cases delusions and hallucinations of sight and hearing occur.

Chronic Starvation: The changes are constant and develop in a constant order. (1) Loss of well-being, hunger, and hunger-pains. (2) Mental and physical lethargy and easy fatigue. (3) Progressive loss of weight which is rapid in the first six months. (4) Polyuria. (5) Increasing wasting, the body weight is reduced by about 40% of the normal. Pigmentation and anaemia. (6) Hypothermia, peripheral vascular stasis in cold and hypotension. (7) Extreme lethargy, gross mental retardation and loss of self-respect. (8) Oedema, first in the feet and lower limbs. (9) Reduced resistance to infection causing diarrhoea, dysentery, tuberculosis, etc.

Cause of Death: Death occurs from exhaustion, circulatory failure due to brown atrophy of the heart or intercurrent infection. Dehydration and hypothermia contribute to death.

Fatal Period: If both water and food are completely withdrawn, death occurs in 10 to 12 days. If food alone is withdrawn, death occurs in 6 to 8 weeks or even more. **Death usually occurs when about 70 to 90% of body fat, and 20% of body protein are lost.**

P.M. Appearances: Rigor mortis sets in and disappears early. Trophic skin changes are common. The hair is dry, lustreless and brittle and nails are also brittle. The face is pale, the skin inelastic and pigmented. Subcutaneous patches of oedema are seen around the ankles, inside the thighs, under the chin, cheeks and eyelids. Ascites is present in ten per cent of cases. **All organs and tissues show changes similar to premature senility. There is extreme emaciation and general reduction in size and weight of all the organs except the brain,** which is sometimes pale and soft. **Fat is almost completely absent in the subcutaneous tissues and also in the omentum, mesentery and about the internal organs, which is never seen in wasting disease.** The fat of the female breast and of the orbit is spared till late. Muscles are atrophied and darker due to increase in lipochrome.

Blood volume is greatly reduced and there is marked anaemia. The heart is small from brown atrophy, and the chambers are empty. The lungs are pale and collapsed and exude very little blood when cut. The liver is atrophied and may show necrosis due to protein deficiency. The gallbladder is distended with bile. Spleen is shrunken. The kidneys show atrophy of the nephron. The urinary bladder is empty. The stomach and intestines show atrophy of all coats and the mucosa is stained with bile. The walls of intestine appear like tissue paper with atrophy of mucosa. The bowel contains offensive fluid and gas. There may be superficial but extensive non-specific ulceration of the bowel like those seen in ulcerative colitis. In children, the skeleton shows spinal curvature, rickets and dental defect. In adults, progressive demineralisation and osteomalacia are seen. Stress fractures may occur. There may be evidence of some intercurrent disease.

Circumstances of Death

Suicide: Sometimes, persons voluntarily abstain from food, for the purpose of exhibition. Lunatics and hysterical women may resort to fasting. It may be resorted to by persons to attract public attention, e.g., fast unto death, for the redressal of grievances.

Homicide: The victim is usually an infant, or any other helpless person, e.g., aged or feeble-minded. Illegitimate children are frequently starved to death. Children starved by their parents or guardians is known as **"baby-farmer"**.

Accident: Accidental starvation may occur during famine, being trapped in pits or mines, landslides, shipwreck, etc. It may be due to ignorance, which leads to a failure to provide enough food or to provide food of right kind. It may also occur in stricture or cancer of the oesophagus or ankylosis of the jaw.

CHAPTER

13 Mechanical Asphyxia

Mechanical asphyxia is a broad term in which enough external pressure is applied to the neck, chest or other areas of the body, or the body is positioned in such a way that respiration is difficult or impossible (Fig. 13-1).

HANGING

Hanging is that form of asphyxia, which is caused by suspension of the body by a ligature which encircles the neck, the constricting force being the weight of the body. The whole weight of the body is not necessary, and only a comparatively slight force is enough to produce death.

Classification: (I) **Depending on degree of suspension:** (a) **Complete hanging:** Body is completely suspended without any part of the body touching the ground. (b) **'Partial hanging':** The bodies are partially suspended or in a sitting, kneeling, lying down, prone, or any other posture with only the head and chest off the ground. The weight of the head acts as the constricting force. (II) **Depending on position of the knot:** (a) **Typical hanging:** The ligature runs from the midline above the thyroid cartilage symmetrically upwards on both sides of the neck to the occipital region and the knot is over the central part of back of neck. (b) **Atypical hanging:** The knot is anywhere other than on the occiput, i.e., on the right or left side or front of the neck.

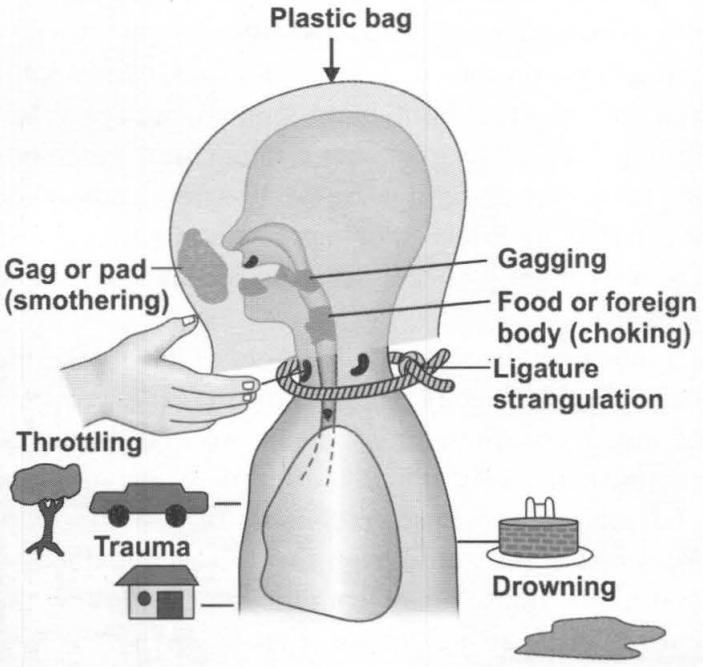


Fig. (13-1). Mechanical asphyxia.

Ligature: A suicide will use any article which is readily available for the purpose, like a rope, metallic chains and wires, leather strap, belt, bedsheet, *scarf, dhotie, saree, turban, sacred thread,* etc.

SYMPTOMS: The first symptoms are loss of power and subjective sensations, such as flashes of light and ringing and hissing noises in the ears. There is intense mental confusion, followed by loss of consciousness (within 15 seconds when a rope is used). Convulsions occur. The face is distorted and livid, eyes prominent, and there is violent struggling. Heart may continue to beat for 10 to 15 minutes after stoppage of respiration.

CAUSES OF DEATH: (1) Asphyxia: The constricting force of the ligature causes compressive narrowing of the lumens of larynx and trachea and forces up the root of the tongue against the posterior wall of the pharynx, and folds the epiglottis over the entrance of the larynx to block the airway. A tension of 15 kg. on ligature blocks the trachea. **(2) Venous congestion:** The jugular veins are blocked by the compression of the ligature which results in stoppage of the cerebral circulation. This occurs if ligature is made up of broad and soft material which cannot sink into tissue to any depth. The jugular veins are closed by a tension in the rope of 2 kg. **(3) Combined asphyxia and venous congestion:** This is the commonest cause. **(4) Cerebral anaemia:** Pressure on the large arteries on the neck produce cerebral anaemia and immediate coma. Death will be slow and asphyxial features are less marked. This occurs with ligature made of thin cord which sinks deeply into tissues. A tension of 4 to 5 kg. on ligature blocks carotid arteries, and 20 kg. the vertebral arteries. This is much less common than venous occlusion.

(5) Reflex vagal inhibition from pressure on the vagal sheath or carotid bodies. **(6) Fracture or dislocation of the cervical vertebrae**. All of these causes may act independently or in concert.

Delayed Death: Death delayed for several days is rare. Delayed deaths occur due to (1) aspiration pneumonia, (2) infections, (3) oedema of lungs, (4) oedema of larynx, (5) hypoxic encephalopathy, (6) abscess of brain, (7) infarction of brain, (8) cerebral softening.

Fatal Period: Death occurs immediately if the cervical vertebrae are fractured or if the heart is inhibited; rapidly if cause is asphyxia, and least rapidly if coma is responsible. The usual period is 3 to 5 minutes.

Postmortem Appearances: External: The ligature mark in the neck is the most important and specific sign of death from hanging (Plate 17-100 to 17-102). Ligature mark on the neck depends on: **(1) Composition of ligature:** The pattern and texture is produced upon the skin, e.g., if thick rope is used, its texture may be impressed in the form of superficial abrasion. **(2) Width and multiplicity of ligature:** When ligature is narrow, a deep groove is made. A broad ligature will produce only a superficial mark. If the ligature is passed twice round the neck, a double mark, one circular and the other oblique may be seen. **(3) The weight of the body suspended and the degree of suspension:** Heavier the body and greater the proportion of the body suspended, the more marked is the ligature impression. **(4) The tightness of encircling ligature:** The ligature impression is deeper opposite the point of suspension. **(5) The length of time body has been suspended:** Longer the suspension, deeper is the groove. **(6) Position of the knot:** The main force applied to the neck by ligature is opposite to the point of suspension. If the point of suspension is in occipital region, front of the neck is involved. If in front, the depth of the groove is limited posteriorly by cervical spine. **(7) Slipping of ligature during suspension:** Frequently, only the portion adjacent to the knot moves. There is a tendency for the ligature to move upwards, this being limited by the jaws. The upward movement may produce double impression of ligature. The lower mark is usually very superficial.

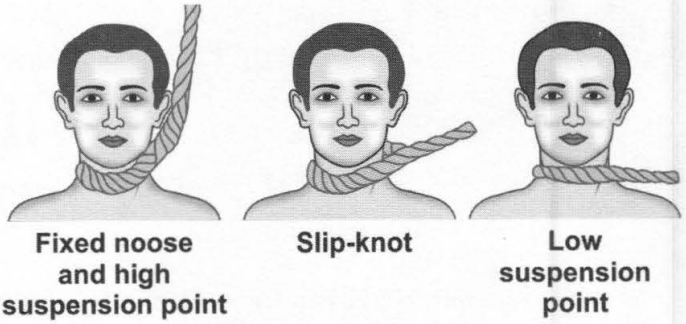


Fig. (13-2). Types of suspension of the body in hanging.

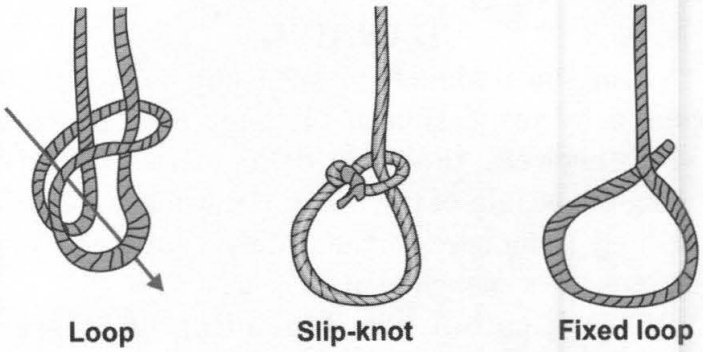


Fig. (13-3). Types of ligature.

Knot: A slip knot (running noose) is produced by attaching a rope to itself creating a loop which can be tightened to produce a running noose. **A binding knot** is a knot in which rope passes at least once around the neck, and is held in place by two ends of the rope being knotted together. This produces a **fixed noose (Fig. 13-3). Reef knot** is a flat knot used in tying reef points consisting of two loops passing symmetrically through each other that will not slip. **Granny knot** is a knot like reef knot, but asymmetrical, apt to slip or jam. **Noose** is a loop with running knot which ties the firmer the closer it is drawn. It is frequently in the form of a simple slip-knot to produce a running noose fixed by granny or reef-knot; occasionally a simple loop is used.

The knot is usually on the right or left side of the neck, ligature usually rising behind the ear to the point of suspension. Sometimes, the knot is in the occipital region and rarely under the chin. In the majority of hangings, the ligature consists of a single loop. Removal of the noose from the deceased is done by cutting the noose away from the knot, and tying the cut ends with string or wire **(Fig. 13-2)**.

Description of Ligature Mark: The ligature produces a furrow or a groove in the tissues which is pale in colour, but it later becomes yellowish or yellow-brown and hard like parchment, due to the drying of the slightly abraded skin. **The course of the groove depends on whether a fixed or running noose has been used. A fixed noose is one in which the rope is knotted (granny or reef knot). A running noose is one in which the end of the rope is passed through the loop (slip knot)** (Fig. 13-3).

Fixed Loop: (1) When the loop is arranged with a fixed knot, the course of the mark is deepest and nearly horizontal on the side opposite to knot but as the arm of the ligature approaches the knot, the mark turns upwards towards it. This produces an inverted V at the site of the knot, the apex of the V corresponding with the site of the knot (Figs 13-4 to 13-6). In the case of a **fixed loop with a single knot in the midline at the back of the head**, the mark is seen on both sides of the neck, and is directed obliquely upwards towards the position of the knot over the back of the neck. (2) In the case of a **fixed loop with a single knot in the midline under the chin**, the mark is seen on the back and both sides of the neck and is directed obliquely forwards towards the position of the knot over the front of the neck. (3) In the case of a **fixed loop with the knot in the region of one ear**, the mark differs on each side of the neck. On the side of the knot, the mark is directed obliquely upwards to the knot, and on the other side it is directed transversely.

Horizontal mark: (1) **When a running noose is applied**, the weight of the body will cause the noose to tighten in a mainly horizontal position. The mark is seen on both sides of the neck, and is usually directed transversely across the front of the neck resembling that of a ligature mark in strangulation, except that it is likely to be seen above the level of thyroid cartilage. (2) **If a running noose fails to tighten**, the mark may resemble one produced by a fixed loop. (3) **In hanging from a low point of suspension**, the groove produced by the ligature is less well marked, and may be at about the level of the upper border of the larynx and more horizontal. (4) **In partial hanging** when the body leans forward, a horizontal ligature mark may be seen.

The ligature mark usually encircles the entire neck, except for the place where the knot was located. A slip knot may cause the noose to tighten and squeeze the skin through the full circumference of the neck. The firmer muscular tissues at the back of the neck do not show clear and deep grooves as are seen at the front or sides of the neck.

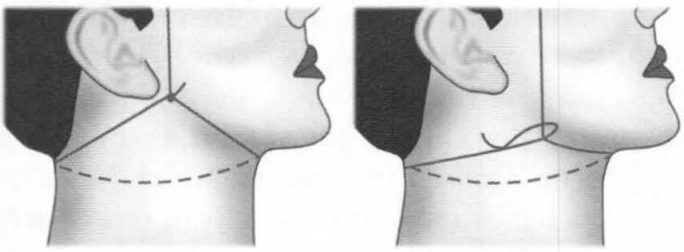


Fig. (13-4). The position of the ligature mark on the neck.

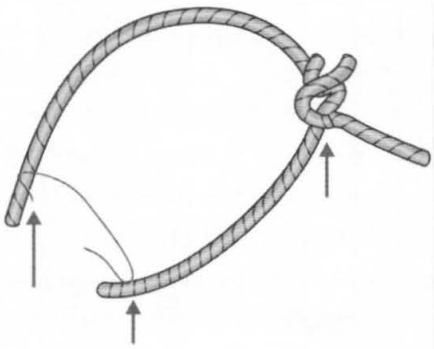


Fig. (13-5). Method of preservation of noose in hanging and strangulation.

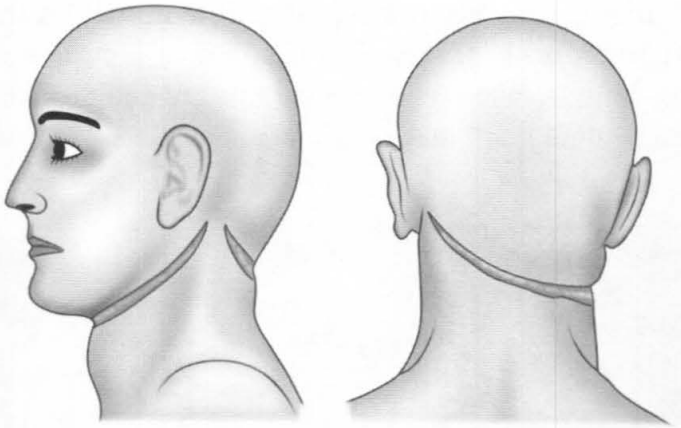


Fig. (13-6). Ligature mark in hanging. Point of suspension is just posterior to the left ear showing inverted V furrow.

Features: The mark is situated above the level of thyroid cartilage between the larynx and the chin in 80% of cases. It may be at the level of the cartilage in about 15%, and below the cartilage in about 5% cases, especially in partial suspension. **The width of the groove is about, or slightly less than**

width of the ligature. When a folded cloth has been used, there may be great difference between the appearance of the neck mark and the size of the ligature. When cloth is pulled tight, certain parts of it become raised into ridges, which form the ligating surface and only these may be reproduced on the skin. When nylon, silk or terylene clothes are used, they may leave a mark only 2 to 3 mm. wide. A loop made of soft material, e.g., towel, scarf, etc. may not produce a ligature mark but the knot may produce an abrasion due to its firmness. Rarely, a narrow wire may be used (cheese-cutter method). Along the edges of the depression, a thin line of congestion or haemorrhage will be seen above and below the groove at some point, usually the deepest, if not throughout its course. **Ecchymoses alone have no significance as to whether hanging was caused during life or not, but abrasions with haemorrhage are strongly suggestive of suspension taking place during life.** Any well-defined pattern in the ligature is likely to be produced in the groove permitting a match of patterns. When fresh, the ligature mark is less clear, but becomes prominent after drying for several hours. **Hanging may occur without visible marks on the victim's neck.** If there is a beard, or if a portion of clothing is caught between the ligature and the skin, no ligature mark may be found under it.

The ligature mark of hanging may be reproduced if the body is suspended within two hours after death, or possibly longer. It can also be produced by dragging a body along the ground with a cord passed round the neck soon after death. **The ligature mark may disappear after several hours following removal of the ligature. Decomposition obliterates the ligature mark.**

Partial Hanging: Hanging may occur simply by leaning against the noose secured to a chair or doorknob, the leg of a table, a bedpost or rail, or the handrail of a stair case, which is slightly higher than the position of the head, the deceased being in a kneeling position, or slumped back or forward and lie prone with only the face and chest off the ground. In these cases, only the weight of chest and arms can cause fatal pressure on the neck **(Figs 13-7 to 13-10). In these cases, the constricting force is**

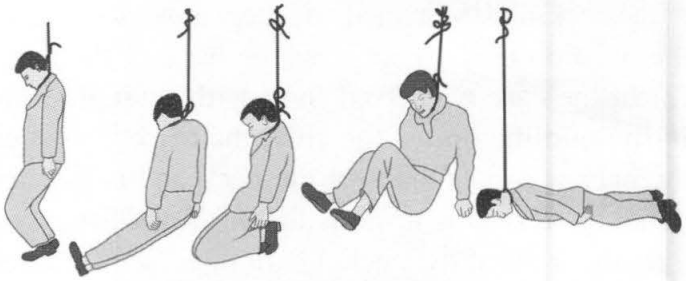


Fig. (13-7). Partial hanging.

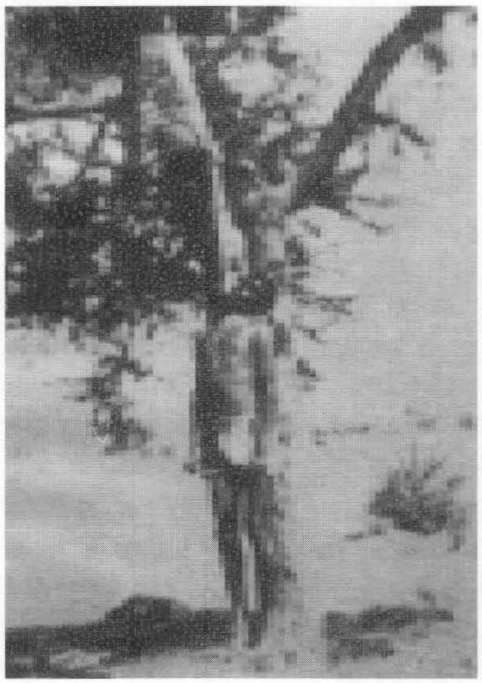


Fig. (13-8). Suicidal hanging.

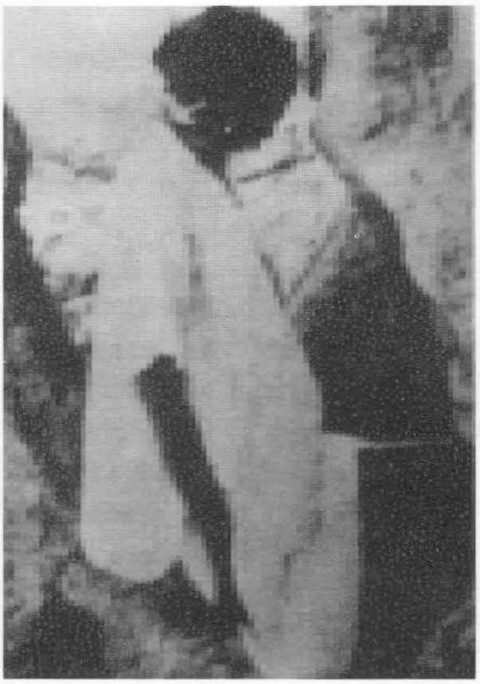


Fig. (13-9). Partial hanging (sitting position).

Fig. (13-10). Partial hanging (kneeling position).

less and congestive changes are more marked, and petechiae are seen, because carotid arteries and jugular veins are occluded, but vertebral arteries continue to supply blood to the head. Bradycardia and fibrillation may cause death when suspension point is very low.

Asphyxial Signs: The signs of asphyxia are most marked in cases in which the noose was placed high up on the throat. Slow asphyxia is the exception in hanging, and is likely to occur only when the point of suspension is a low one, or ligature exerts pressure below the chin and does not encircle the neck. When suspension is complete or in the standing posture, asphyxial signs are minimal, and petechial haemorrhages are less common. In incomplete suspension, asphyxial process is slow, face dusky-purple, congested and often swollen.

Other Signs: In death due to vagal inhibition or injury to spinal cord, face will be pale. If the body has been suspended for some time, postmortem lividity is seen in the lower limbs and hands, while the upper part of the body will be pale. Petechial haemorrhages may be found in the skin of the legs. The neck is stretched and elongated and the head is always inclined to the side opposite to the knot. The face is usually pale, but is sometimes congested and swollen, due to leakage of oedema fluid into the soft tissues, if the veins were constricted before the arteries. Majority of victims of hanging are seen with pale faces. The eyes are frequently protruded and firmer and conjunctivae usually congested. The eyes are closed or partially open and the pupils are usually dilated. **If the ligature knot presses on cervical sympathetic, the eye on the same side may remain open and its pupil dilated** ***(le facie sympathique).*** **It indicates antemortem hanging.** The tongue is usually swollen and blue especially at the base, and usually forced against the teeth when the jaw is shut, or the tip may be found projecting between the lips. The lips, and the mucous membrane of the mouth are blue. **Saliva may be found dribbling from the angle of mouth** when the head is drooping forward. This is due to the increased salivation before death due to the stimulation of the salivary glands by the ligature. Slight haemorrhage or bloody froth is sometimes seen at the mouth and nostrils. The hands are clenched, especially in violent hanging. Engorgement of the penis with blood occurs from hypostasis; it may be semi-erect, and semen may be found at the tip. Urine and faeces may escape due to relaxation of the sphincters.

Internal: The neck should be examined after removal of the brain and viscera from the chest and abdominal cavities. The **tissues under the mark are dry, white and glistening with occasional ecchymoses in the adjacent muscles.** The muscles of the neck, especially the platysma and sternomastoid are ruptured (5 to 10%) if violence has been considerable. **Haemorrhages in strap muscles are present in about 25%.** In some cases (5 to 10%) the intima of the carotid arteries show transverse splits (under the groove, usually around the region of sinuses), with extravasation of blood in their walls due to stretching and crushing. The vertebral arteries show ruptures, intimal tears, and subintimal haemorrhages in some cases. Injury to the trachea is unusual. Petechial haemorrhages may be found on epiglottis, in the larynx and trachea. The trachea is usually congested. The lungs are congested, oedematous and exude bloody serum on section in cases of constriction occurring at the end of expiration; but they are pale if constriction occurred at the end of inspiration. Subpleural ecchymoses may be found. The abdominal organs are usually congested. The brain is usually normal. Subarachnoid effusions are common.

Fracture of hyoid bone and thyroid cartilage: Opinion varies regarding the frequency of fracture of the hyoid bone. **Estimates range from 0 to 60%, but the average is 15 to 20%. Fractures are rare below 40 years,** because of the elasticity of the cartilage and mobility of the joints. **The fracture is common in persons above 40 years and involves the great horns, at the junction of inner two-thirds and outer one-third. The superior horns of the thyroid may be fractured in about 40% of cases above 40 years.**

Diagnosis: (1) Ligature mark around the neck, (2) presence of abrasions, ecchymoses and redness about the ligature mark, (3) trickling of saliva from the mouth, (4) ecchymoses of the larynx or epiglottis, (5) rupture of the intima of the carotid, and (6) postmortem signs of asphyxia.

The Circumstances of Death

ACCIDENTAL HANGING: (1) It is seen in children during play while imitating judicial hanging, or in athletes who are in the habit of exhibiting hanging. Some padding between ligature and neck suggests accident. (2) Workmen in falling from scaffolding may be hanged by becoming entangled in ropes. (3) When boys climb trees or railings, they may lose their foothold and in falling, some garment is caught by tree branch or bar and is drawn tight round the neck. (4) A person who slips when descending a ladder may be suspended by one of its rungs, or a slip on a staircase may result in suspension on the edge of one of the treads. (5) It may be associated with abnormal sexual behaviour.

SUICIDAL HANGING: Hanging is a common method of committing suicide. In most cases, the body is found in a completely suspended position, with the ligature tied to a beam, nail, hook, window-casing, branch of a tree, etc. (Plate 17-98 and 17-99). The body must be in a position compatible with self-suspension. Complete suspension of the body in the absence of any platform is unusual in suicide. Unusual positions, e.g., where the parts of the body touched the ground, kneeling or reclining, are almost diagnostic of suicide. The hands and feet are sometimes tied by the victim before hanging himself. The position of the ligature with reference to the knot, and the manner in which it is attached to the support must be compatible with self-suspension. Blindness or age do not prevent hanging. A person with tracheostomy can commit suicide by handing, as death occurs due to vagal inhibition. Sometimes hanging is adopted as a last resort after other forms of suicide, e.g., cutting the throat or wrists, stabs, firearm wounds, ingestion of poison, etc., have failed to produce death. Suicide pacts affected by hanging are rare.

HOMICIDAL HANGING: It is extremely rare. It is difficult for a single assailant to carry it out unless the victim becomes unconscious by injury or by a drug, or is taken unawares, or is a child or a very weak person. Homicide should be suspected: (1) Where there are signs of violence or disorder of furniture or other objects. (2) Where the clothing of the deceased is torn or disarranged. (3) Where there are injuries, either offensive or defensive. In all doubtful cases, circumstantial evidence is important.

SUSPENSION OF BODY AFTER DEATH (POST-MORTEM HANGING): A person may be murdered and the dead body suspended to simulate suicide. Look for signs of dragging to the place of suspension. When a dead body is suspended, the rope is usually tied first around the neck, and then around the beam, branch of a tree, etc. The beam shows evidence of rope having moved from below upwards as the body has been pulled up. In true suicidal hanging, the rope moves from above downwards. Further, fibres from the rope may be found on the hands of the victim in suicidal hanging but not in case of postmortem hanging. In most cases, the internal signs are clearly not those of hanging, although in most cases ligature mark cannot be distinguished.

Lynching: The name is derived from captain William Lynch, who used to order hanging on the spot without trial in U.S.A., where a black rapist was used to be lynched by angry white mob, with a view to teaching others a lesson for committing an offence. **It is homicidal hanging. Sometimes a person suspected to be involved in murder, rape, dacoity, etc. is overpowered by a mob and is hanged forcibly from a tree, etc.**

JUDICIAL HANGING: Legal death sentence is carried out by hanging the criminal. The face of the person is covered with a dark mask, and he is made to stand on a platform above trapdoors, which open downwards when a bolt is drawn. A rope to allow a drop of 5 to 7 metres according to the weight, age and build of the person is looped round the neck, with the knot under the angle of the jaw. On drawing the bolt, the person drops to the length of the rope. **The sudden stoppage of the moving body associated with the position of the knot, causes fracture-dislocation at the level of the second and third, or third and fourth cervical vertebrae.** Bilateral fractures of either the pedicles or laminae of the arch of the second, third or fourth cervical vertebrae may occur (**hangman's fracture**). **The upper cervical cord is stretched or torn across. It results in immediate unconsciousness,** but heart beats and respiratory movements may continue up to 10 to 15 minutes, and spasmodic muscular jerking may occur for a considerable time. **Asphyxial signs are not seen in properly performed judicial hangings.**

STRANGULATION

Strangulation is that form of asphyxia, which is caused from constriction of the neck by a ligature without suspending the body.

Types: (1) Strangulation by a ligature. (2) Manual strangulation or throttling. (3) Garroting. (4) Mugging. (5) Bansdola.

Cause of Death: Death may be due to (1) asphyxia, (2) cerebral anoxia or venous congestion,

(3) combined asphyxia and venous congestion, (4) vagal inhibition.

Ligature: The ligature may consist of a wide variety of objects such as cords, wires, ropes, belts, scarves, ties, towels, stockings, bed linen, etc.

Postmortem Appearances: (A) External: The Ligature Mark: It is usually a well-defined and slightly depressed mark at any level on the neck, but usually about the middle or below the thyroid cartilage. The mark completely encircles the neck transversely, but is more prominent at the front and sides than at the back. The skin of the front of the neck is more likely to be damaged by a ligature than the thicker, tougher skin at the back of the neck. If a knot has been applied, there may be a wider area of bruising at the site of the knot. **If the ligature has been tightened by pulling on the crossed ends, the marks produced by the two ends will be at different levels.** Multiple turns produce a complex mark, in which it may be possible to trace the number of turns, but a complex ligature composed of several pieces knotted together may produce a mark which simulates multiple turns, though there was only one.

Rarely a narrow wire may be used, the so-called "cheese-cutter" method. When a narrow ligature, e.g. a cord, wire or thin rope is used, the ligature mark is slightly narrower than the actual diameter of the ligature itself. When the ligature is still in position when the body is examined, it may appear to be deeply embedded in the skin, and on removal a deep groove may be seen in the skin. This embedding may be increased by oedema of the tissues, especially above the ligature, which initially may not have been applied so tightly. Strong pressure may lacerate the skin or cut into the deeper tissues and cartilages. **When a folded cloth has been used, there may be great difference between the appearance of the neck mark and the size of the ligature.** When fabric is pulled tight, certain parts of it become raised into ridges which form the ligating surface, and only these may be reproduced on the skin. **When nylon, silk or terylene fabrics are used, they may leave a mark only 2 to 3 mm. wide. The mark may be oblique as in hanging, if the victim was sitting and the assailant applied ligature on the neck while standing behind him, thus using the force backwards and upwards, or if the victim has been dragged by a cord after he has been strangled in a lying down posture.** The base of the mark is usually pale. There may be exudation of tissue fluid which later dries, forming a stiff film. **Petechial haemorrhages are usually found immediately adjacent to the ligature mark, which is a confirmation that the mark was made during life.** Reddening and congestion is also common immediately above and below the groove, due to squeezing out of the blood by the pressure. This does not necessarily indicate that the ligature was applied during life. **The base becomes dry, hard and parchment-like several hours after death if the skin has been abraded.** The pattern of the ligature may indicate the agent, e.g., a woven belt, plaited electric wires, links of chains, spiral rope patterns, etc. If a rough ligature, e.g., a rope is used, and if there is some movement of the rope on the skin during a struggle, the skin may show marked abrasions and haemorrhages, and the deep muscles of the neck are grossly bruised. A ligature mark may be interrupted at the front by the presence of the clothing or by the victim's fingers, in an attempt to pull the ligature away. If the ligature used is soft and yielding, e.g., a towel, scarf, etc., and if it is removed soon after death, the mark in the neck may be very slight or absent. The deep muscles of the neck may show little or no bruising. The **ligature mark of strangulation is not obliterated by putrefaction, but is better preserved than the skin beyond it because of compression. Even if the mark is obscured, subcutaneous haemorrhages in relation to the mark may be found.**

Description of ligature mark: Depth, width, direction, raised ridges of skin, zones of hyperaemia, presence of duplicate strangulation marks, dried excoriations due to slippage of the implement, marks due to textile weave pattern and structure, distribution of petechiae in the skin, bruising, scratch marks, blisters in the strangulation mark should be noted.

Pseudo-strangulation: Occasionally, depressed marks are seen on the dead bodies of infants and children, in whom the neck is short. (1) These marks are produced from folds in the skin due to bending of the head. (2) A similar mark of depression is found in short-necked persons after death, across front of the neck. (3) It can also be seen in decomposing bodies

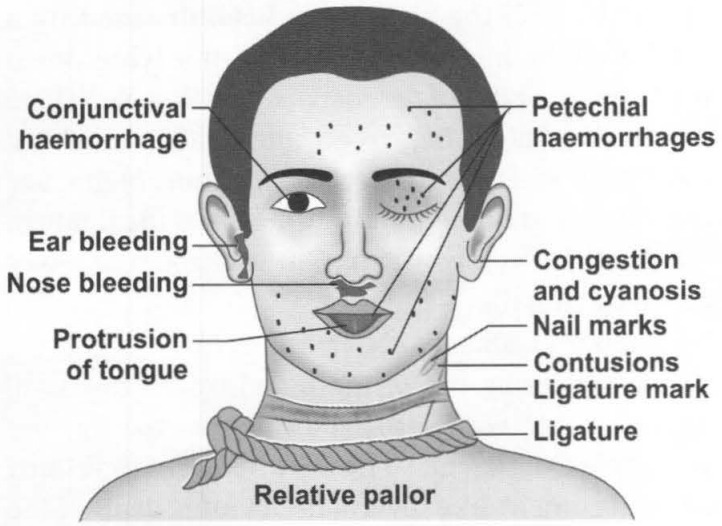


Fig. (13-11). Signs of strangulation.

with tight collars, buttoned shirt at the neck, or other clothing round the neck. In these cases, a deep groove simulating ligature mark of strangulation is produced due to the swelling of the tissues around the tight-fitting garment, as the body decomposes.

Signs of Asphyxia: If there is slight vagal effect and some venous constriction, there will be slight congestion of head and neck and occasional petechial haemorrhages. With moderate venous constriction and some respiratory obstruction asphyxial signs are moderate. When constricting force is great, these signs are marked. Intense congestion and deep cyanosis of the head and neck is seen. The face may show bluish patches and is swollen, the eyes wide open, bulging and suffused, the pupils dilated, the tongue swollen and often bruised, dark-coloured and protruded. Petechial haemorrhages are common into the skin of the eyelids, face, forehead, behind the ears and scalp. Blood-stained froth may escape from the mouth and nostrils and there may be bleeding from the nose and ears (**Fig. 13-11**). The hands are usually clenched. The genital organs may be congested and there may be discharge of urine, faeces and seminal fluid. These external signs may be absent if death occurs quickly from vagal inhibition, due to pressure on carotid sheath. A sudden compression of the wind-pipe causes almost immediate unconsciousness and death.

Internal: There may be superficial haemorrhages under the ligature mark, though this is often minimal. There is severe congestion and haemorrhage into the tissues in and above the area compressed. The adjacent muscles of the neck are usually lacerated. **In some cases, no external mark is seen on the surface of the neck but extensive bruising of the deeper tissues may be found.** The extent of the bruising depends upon the degree of pressure used. **A ligature which is tightly applied on the neck until death occurs, may fail to produce any bruising.** The intima of carotid arteries is not usually damaged, but a deeply sunken narrow ligature applied with force may damage the carotid arteries.

Bruising of the root of the tongue and floor of the mouth may occur. Haemorrhage may be seen under the mucosa of the larynx, especially beneath the vocal cords. These haemorrhages are larger than petechial haemorrhages of asphyxia. The larynx, trachea and bronchi are congested and contain frothy, often blood-stained mucus. **Subcapsular and interstitial thyroid haemorrhages are common.** The lungs are markedly congested and show ecchymoses and larger subpleural haemorrhages. **Silvery-looking spots under the pleural surface due to rupture of the air cells, which disappear on pricking, are seen in more than fifty percent cases.** Pulmonary oedema may be present. The parenchymatous organs show intense venous congestion, and in young person ecchymoses are usually seen on the heart and kidneys. The brain is congested and shows petechial haemorrhages. **Hyoid bone may be fractured in older persons in 10 to 15% of cases. Fracture of the thyroid cartilage is more common especially of one or both superior horns.** Fracture of thyroid laminae and cricoid cartilage is less common, and occurs only when considerable force is used, or if some solid object is included in the ligature and applied over the front of the neck. Trachea is rarely broken.

MEDICOLEGAL QUESTIONS: (1) WHETHER DEATH WAS CAUSED BY STRANGULATION? In death due to strangling, the general features of asphyxial death are present. Their local distribution in the head and neck is strongly presumptive of strangulation. This is confirmed by the ligature mark on the neck. Evidence of violent compression or constriction of the neck during life is obtained from the presence of bruising or ecchymoses about the marks on the neck, haemorrhages in the strap muscles, under the skin, in the sides of the tissues around the trachea and larynx,

Table (13-1). Difference between hanging and strangulation

Trait	Hanging	Strangulation by ligature
(1) Ligature mark:	It is oblique, does not completely encircle the neck; usually seen high up in the neck between the chin and larynx. The base is pale, hard and parchment-like.	It is transverse, completely encircling the neck; usually seen low down in neck below the thyroid cartilage. The base is soft and pale or reddish.
(2) Abrasions and ecchymoses:	About the edges of ligature mark not common.	About the edges of the ligature mark is common.
(3) Bruising:	Of the neck muscles less common.	Of the neck muscles more common.
(4) Subcutaneous tissues:	White, hard and glistening under the mark.	Ecchymosed under the mark.
(5) Neck:	Stretched and elongated.	Not stretched or elongated.
(6) Signs of asphyxia:	External signs less marked.	External signs well marked.
(7) Face:	Usually pale and petechiae are not common.	Congested, bluish, and marked with petechiae.
(8) Tongue:	Swelling and protrusion is less marked.	Swelling and protrusion is more marked.
(9) Saliva:	Often runs out of mouth.	Absent.
(10) Bleeding:	From the nose, mouth and ears not common.	From the nose, mouth and ears common.
(11) Involuntary discharge:	Of faeces and urine less common.	Of faeces and urine more common.
(12) Seminal fluid:	At glans is more common.	At glans is less common.
(13) Larynx and trachea:	Fracture rare.	Fracture is more common.
(14) Emphysematous bullae:	Not present on the surface of the lungs.	Very common on the surface of the lungs.
(15) Carotid arteries:	Damage may be seen.	Damage is rare.
(16) Thyroid cartilage:	Fracture is less common.	Fracture is more common.
(17) Hyoid bone:	Fracture is more common.	Fracture is less common.

and in the laryngeal structures themselves. In cases of putrefaction, a medical opinion about strangulation can be given fairly, if there are signs of mechanical violence applied to the neck, e.g., fracture of the larynx or hyoid bone (Table 13-1).

(2) WHETHER THE STRANGULATION WAS SUICIDAL, HOMICIDAL OR ACCIDENTAL?

SUICIDAL STRANGULATION: Suicide by strangulation is rare. Various methods of tightening the ligature are employed by the victims. (1) The ligature is tightened like a tourniquet, but the person can apply a single or double knot, before consciousness is lost. (2) Several turns of rope are tied round the neck with a knot which is usually single and in front or at the side or back of the neck. (3) A cord may be tied around the neck and twisted tightly by means of a stick or some other solid material used as a lever. When consciousness is lost, although grip on stick is released, the ligature will not become loose, as it gets arrested against the shoulder or chin. (4) A running noose is applied to the neck and the free end of the rope to which a weight is attached, is thrown over the end of the bed on which the victim lies. (5) A person may strangle himself by leaning with the whole weight of his body on a cord passed round the neck and attached to a fixed point. In all cases of suicidal strangulation, the ligature should be found in situ, and the body should not show signs of violence or marks of struggle.

HOMICIDAL STRANGULATION: Strangulation is a common form of murder. Many of the victims are adult women, and frequently strangulation is then associated with sexual assault. Usually there is a single

turn of ligature round the neck, with one or more knots (granny or reef-knots) at the front or side of the neck. When there are two or more firm knots, each on separate turns of the ligature, homicide is almost certain. Abrasions are usually seen due to movement of the ligature across the neck. The mark may either completely encircle the neck or may be seen only at the front, when the ligature is pulled tightly from behind. The mark may also be sloping, if the ligature is pulled upwards from behind, and the position is high up at the level of the hyoid bone. Sometimes, a ligature is passed over the body, and then tied to the hands and feet to simulate suicide. In such cases, the manner of tying should be examined. The presence of a complex type of knotting in the cord, e.g., the presence of a reef knot, suggests homicidal strangulation. Evidences of struggle are usually found but if a person is taken unawares, and the ligature is suddenly placed around the neck and pulled tightly, the person loses consciousness quickly and is unable to offer much resistance. Sometimes, circumstantial evidence, such as time, place, locked doors, and windows, motive, etc., is almost the only evidence to suggest either suicide or homicide.

ACCIDENTAL STRANGULATION: (1) Children may get entangled in ropes during play, or the neck may be caught in window cords, etc. (2) Infants are sometimes strangled in their cots when the neck is caught inside bars, in restrainers, braces, etc. (3) Persons under the influence of alcohol, epileptics, and imbeciles may be strangled either by a tight scarf or collar and neck tie. (4) It may occur if an intoxicated person rests the neck against a bar or other hard object. (5) It may occur when a string used in suspending a weight on back, slips from across the forehead and compresses the neck. Accidental strangling may occur in uterus when the movement of the foetus causes the umbilical cord to encircle the neck.

Ligature marks produced after death do not show bruising. Either a grooved impression is seen on the skin which is not injured, or yellow or brown abrasion without signs of vital reaction.

THROTTLING OR MANUAL STRANGULATION

Asphyxia produced by compression of the neck by human hands is called throttling. Death occurs due to occlusion of carotid arteries or vagal inhibition which is much more common than in ligature strangulation, hanging excepted.

Postmortem Appearances: External: Neck injuries: The situation and extent of the contusions on the neck will depend upon: (1) the relative positions of the assailant and victim, (2) the manner of grasping the neck, and (3) the degree of pressure exerted upon the throat. **The bruises are produced by the tips or the pads of the fingers. Their shape may be oval or round and of the size of the digits, but continued bleeding into the contused area usually increases the size:** (1) A grip from right hand from the front produces a thumb impression on the right side of the victim's neck, which is usually under the lower jaw over the cornu of thyroid. Several fingermarks are seen on the left side of the neck obliquely downwards and outwards, and one below the other, but sometimes are grouped together and cannot be distinguished separately. (2) In a grip from behind the victim, the pressure is applied all-round the neck, but some areas of bruising are more prominent due to the pressure of the fingertips. (3) When both hands are used to compress the throat, the thumb-mark of one hand and the finger-marks of other hand are usually found on either side of the throat. Sometimes, both thumb-marks are found on one side and several finger-marks on the opposite side. (4) A grip from both hands, one being applied to the front and the other to the back, produces bruises on the front and back of the neck. **Due to the shifting of the grip, and sometimes the frank struggle of victim, bruises may be seen in a completely haphazard manner. When fresh, the bruises are soft and red, but after several hours they appear brown, dry and parchment-like.**

Fingernail abrasions: They are of two types: (1) When the pressure is static, regularly curved (crescentic), comma-like, dash-like or straight, up to 1.5 cm. in length and a few millimetres in width. Nails with straight border give unpredictable results. As the nails become more pointed towards the centre of the free border, paradoxical results are more common. Fixation of the skin at the centre occurs where the nail digs in with escape at the margins, so that when the traction releases, the mark appears inverted, with the convexity facing the finger pad. (2) When the nails skid down the skin, parallel, linear lines several cm. in length may be seen.

Bruising may not be seen: (1) If a soft material is kept between the hand and throat, (2) If pressure upon the neck is maintained until after the death of the victim, because there has been no blood flowing to extravasate from the damaged vessels. (3) If the

victim is unconscious and amount of pressure to the neck is minimal.

The tongue may or may not be bitten, but is usually protruded. Marks of struggle and external appearances of asphyxia are similar to those found in strangulation. Congestive asphyxial signs appear in 15 to 30 seconds of pressure on the neck, but if a change in grip occurs and fingers impinge on carotid structures, reflex cardiac arrest occurs, so that the intensity of congestive changes may be of any degree in any given death.

Neck dissection: To obtain a bloodless field in the neck, the abdominal and thoracic organs should be removed, the head opened and the brain removed, and then head is moved slowly up and down and allowed to drain of blood. The neck structures should be dissected *in situ.*

Internal: The soft tissues of the neck are compressed and forced upwards and backwards against the cervical vertebrae. **Bruises may be found in the deep layers of skin, in the superficial and deep fascia, on the sheaths of the muscles, and in the substance of the thyroid gland. The bruises are usually separate, and usually the muscles are involved. In some cases, bruising may be absent externally, although the deeper tissues may show fairly extensive bruising.** Deep haemorrhages may be present in the base of tongue, usually at the sides. Deep congestion is seen in central or whole posterior part of tongue. The marks on the surface of the neck may not exactly correspond with the internal bruising, because of the mobility of the skin which may alter the relationship which normally exists between it and underlying structures. The extent of bruising depends upon the degree of pressure used. In some cases, a small area of bruising on either side of the midline and usually over the thyroid cartilage or the hyoid bone may be the only finding. Sometimes, the bruising of the muscles is very slight, and as such the muscles of the neck should be dissected individually *in situ*. The muscles surrounding the larynx, both anteriorly and posteriorly frequently show well marked bruising. The sternomastoid and deeper strap muscles may show patches of bleeding or even frank haematoma. **Muscles, especially the sternomastoid may be torn.** The intima of carotid arteries show rupture in 10 to 15% cases at or near the carotid sinus. Haemorrhages may occur in pharynx, tonsils, base of the tongue and upper part of the larynx. The lungs show congestion with subpleural petechial haemorrhages. Pulmonary oedema may be present. A fine froth, often bloodstained is seen in the bronchi. The brain is usually congested and may show petechial haemorrhages in white matter. Subarachnoid haemorrhages and rarely areas of small haemorrhages in the brain may be present. Other internal features are similar to that of strangulation. Bruises of the laryngeal mucous membrane is frequent, sometimes accompanied by severe laryngeal swelling.

Fractures: The **larynx is usually squeezed, and fractures are common especially in old persons with calcified cartilages. Fracture of the superior cornu of the thyroid cartilage at its base is common** due to the local pressure, but the extent of damage varies. **Often, only the right horn may be broken to which the thumb was applied.** Fractures of the body of the thyroid cartilage are rare. The body of the thyroid cartilage can be broken by a **karate blow** to the front of the neck or by contact with the handle bar of a bicycle or the edge of a chair or any projecting object. **Fractures of the hyoid bone with inward driving of its distal fragments occur in 30 to 50% of cases**, either due to direct lateral compression or due to indirect violence. In throttling, squeezing of the sides of the hyoid bone and larynx causes the fractured margins to point outwards. Fracture of the cricoid cartilage is seen only in fatal cases and occurs due to the anteroposterior compression of it against the spine, e.g., by the thumbs or the forearm of the assailant. Focal haemorrhage is always seen around fracture produced during life. **If bleeding is not seen by the naked eye, but histology of the fracture site shows some red cell extravasation, this can be accepted as antemortem in the presence of surface bruises, scratches, muscle bleeding, intralaryngeal and tongue haemorrhages.**

Rapid death: Rapid or instantaneous death can occur due to sudden cardiac arrest, when a pale face with no signs of asphyxia is seen. This is commonly seen in throttling, often in hanging and less often in ligature strangulation.

Palmar strangulation: The palm of one hand is placed horizontally across the mouth and nostrils without using the fingertips, its pressure being

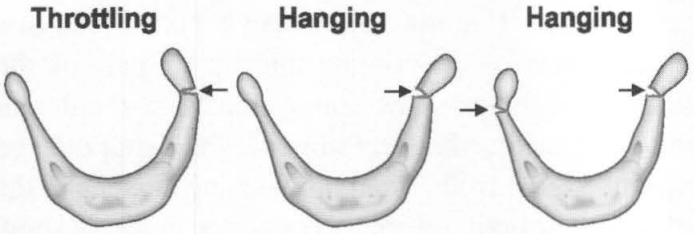


Fig. (13-12). Fracture of the hyoid bone in throttling and hanging.

reinforced by placing the other palm on the top of it at right angles, the heel of the upper palm pressing upon the front of the neck. The face is congested with petechiae. Diffuse bruising with fracture of thyroid cartilage may be seen. In most cases, the victim is intoxicated.

HYOID BONE FRACTURES: They can be classified into three groups.

(1) Inward Compression Fractures: This is seen in case of throttling, where the main force is an inward compression acting on the hyoid bone. **The fingers of the grasping hand squeeze the greater horns towards each other, due to which the bone may be fractured and the posterior fragment is displaced inwards. The periosteum is torn on the outer side of the bone, but not on the inner side (Fig. 13-12).** In such cases, if the body of the bone is grasped in one hand, and the distal fragment between the finger and thumb of the other hand, the distal fragment can be easily bent in an inward direction, but outward movement is limited to the normal position only. At the joint between the greater horn and body of hyoid, a similar fracture may be seen. In some cases, bilateral inward fractures may occur.

(2) Anteroposterior Compression Fractures: In cases of hanging, the hyoid bone is forced directly backwards, due to which the divergence of greater horns is increased, which may fracture with outward displacement of the posterior small fragment. In such cases, the periosteum is torn on the inner side of the fracture only, due to which the fragment can be easily moved outwards, but inward movement is limited to the normal position only. **Fracture may occur, either in the greater horn or at its junction with body, and it may be bilateral. Outward fractures of greater horn of hyoid bone are seen in ligature strangulations, run over motor vehicle accidents, blows on the front of the neck, etc.**

(3) Avulsion Fractures: They **occur due to muscular overactivity without there being direct injury to the hyoid bone.** They are also called "tug" or "traction" fractures.

The cartilaginous separations between the greater horns and the body, and the joints between the lesser horns and the body, or the presence of incomplete bony union of the hyoid bone parts, should not be mistaken for fractures. **A hyoid fracture should not be diagnosed as antemortem in origin, if there is no recent haemorrhage at the fractured site.** The neck should be X-rayed in lateral oblique views to demonstrate fracture of the hyoid and cricoid.

Medicolegal Questions: (1) Whether death was caused by throttling?

The usual diagnostic signs of death due to manual strangulation are: (1) Cutaneous bruising and abrasions. (2) Extensive bruising with or without rupture of the neck muscles. (3) Congestion of the tissue at and above the level of compression. (4) Fracture of the larynx, thyroid cartilage, and hyoid bone. (5) Cricoid cartilage is almost exclusively fractured in throttling. (6) General signs of asphyxia.

(2) Whether the throttling was suicidal, homicidal or accidental?

SUICIDAL THROTTLING: Suicide by throttling is not possible because the compression of the windpipe produces rapid unconsciousness and the fingers are relaxed.

HOMICIDAL THROTTLING: Throttling is a common mode of homicide, because the hand is immediately available. It is a method of choice in infants. The victims are usually infants, children or women. Adults can be throttled when under the influence of drugs or drink, or stunned, or taken unawares. In an adult, signs of struggle are usually present, but if the throat is forcibly grasped suddenly and firmly compressed, the victim cannot struggle. Sometimes, it is preceded by rape or attempted rape. The victim may have been restrained by the throat during intercourse or throttled to stop her cries. If contusions and fingernail abrasions are present on the neck, the presumption must be of homicide.

ACCIDENTAL THROTTLING: A sudden application of one or both hands on other persons throat as a demonstration of affection, in joke, as a part of physiological experiment, etc., may cause death from cardiac inhibition.

GARROTTING: The victim is attacked from behind without warning. The throat may be grasped or a ligature is thrown over the neck and quickly tightened by twisting it with a lever, which results in sudden loss of consciousness and collapse. The assailant is then able to tie the ligature with one or more turns. In this way a single assailant can kill a healthy adult male. This method is usually used in lonely places to kill travellers and to rob them. An iron collar tightened by a screw for strangling **(Spanish Windlass)** as a mode of execution was once employed in Spain.

MUGGING: Strangulation is caused by holding the neck of the victim in the bend of the elbow. Pressure is exerted either on front of the larynx, or at one or both sides of the neck by the forearm and upper arm. The attack is usually made from behind. The postmortem appearances are those of ligature strangulation with a broad object, i.e., the signs are minimal. Internally there may be diffuse bruising, but this may be slight or absent. There may be bruising behind the larynx and in the strap muscles of the neck. In some cases, the neck may be pressed by the foot or knee.

BANSDOLA: One strong bamboo or stick is placed across the back of the neck and another across the front. Both the ends are tied with a rope due to which the victim is squeezed to death. Sometimes, a stick is placed across the front of the neck and the assailant stands with a foot on each end of the stick. If a stick or foot is used, a bruise is seen in the centre of the neck across the windpipe corresponding in width to the substance used. If two sticks are used, a similar mark will be seen on the back of the neck. Sometimes, the chest may be squeezed forcibly between two sticks placed across the back and front of the upper part of the chest.

Compression of the Neck: This is a broad term used for non-specific causes of neck pressure which may be sudden. In hard compression, such as stamping or jumping on the neck, or karate chops, and flying kicks, soft tissues are grossly damaged. Death may be rapid with crush injury and seepage of blood into the tissues, or it may be asphyxial due to swelling or obstruction of the airway. **Soft, prolonged neck compression** occurs in wrestling, carotid sleeper hold and choke hold.

SUFFOCATION

Suffocation is a general term to indicate that form of asphyxia where entry of air to the lungs is prevented by any means other than pressure on the neck or drowning.

Environmental Suffocation: Death from hypoxic hypoxia may result from breathing in a vitiated atmosphere (deficient in oxygen). The concentration of oxygen in air is about 21% and CO_2 0.033%. CO, CO_2, methane, sulphuretted hydrogen and sulphur dioxide are commonly found in vitiated atmosphere. Deaths are almost always accidental. **Suffocation occurs in air-tight place, such as when children become locked in disused refrigerators or large boxes or trunks during play.** It may occur in vicinity of lime kilns or excavations in chalk rock. A person may be suffocated on entering a confined space, such as tanks, grain-bins, silos, deep tanks of a ship, fermenters, tanning vats, unused wells, sewers, etc. In such cases, petechial haemorrhages are absent. Congestion and cyanosis may or may not be present.

SMOTHERING

This is a form of asphyxia which is caused by closing the external respiratory orifices either by the hand or by other means, or by blocking up the cavities of the nose and mouth by the introduction of a foreign substance, such as mud, paper, cloth, etc.

Suicide by Smothering: Suicidal smothering by the hand is impossible. Suicide is possible by burying the face in a mattress or lying against the bed clothing to obstruct the nose and the mouth. It is usually seen in the mental patients or prisoners. Suicidal smothering can be effected by tying a polythene or similar bag over the head.

Accidental Smothering: Most fatal smotherings are accidental. Infants covered with blankets or bedding will not die of smothering. (1) An epileptic or intoxicated person may smother himself accidentally by burying his face in a pillow, or covering with bed clothes. (2) A person may accidentally fall into a large quantity of semi-solid or finely divided material, like mud, ashes, feathers, grain, sand, coal dust, etc., so that his mouth and nose are covered by the substance. The victim may struggle, inhale some of the material into his air-passages and swallow some into his stomach in an effort to breathe. (3) Children may be suffocated accidentally while playing with plastic

bags. (4) **Plastic bags** may be applied to the head for experiment or autoerotic exercise, as partial asphyxia is believed to increase sexual sensation. (5) It also occurs if the membranes remain round the head of the newborn after delivery.

Homicidal Smothering: Homicide is possible where the victim is incapacitated from drink or drugs, very weak, child or old person, in illness, and when the victim is stunned by a blow. Usually the mouth and nose are closed by a hand or cloth, or the face may be pressed into a pillow.

Autopsy: Obstruction by bed clothing, a pillow, a cushion, etc., applied with skill, may not leave any external signs of violence, especially in the very young and the old. The asphyxial signs and symptoms are severe because death usually results due to slow asphyxia. If the orifices are closed by the hand, there may be scratches, distinct nail-marks, or lacerations of the soft parts of the victim's face. The lips, gums and tongue may show bruising or laceration. Blood may ooze out from the mouth and nose. The tongue may be protruded, and may have been bitten. In some cases death is rapid due to reflex cardiac arrest, and asphyxial signs are absent. Signs of asphyxia are faint or absent in case of plastic bag suffocation. Moisture usually collects in the bag.

Internal: Blood-stained frothy fluid is present in air-passages. Slight acute emphysema and oedema of the lungs with scattered areas of atelectasis, petechiae and congestion are the major findings. The internal organs are deeply congested and sometimes show small haemorrhages. **If a person is buried alive, earth and sand will be found in the respiratory tract.**

GAGGING: This is a form of asphyxia which results from pushing a cloth or soft object into the mouth, or from the closure of mouth and nose by a cloth or similar material, which is tied around the head. A gag, such as rolled-up cloth pushed into the mouth sufficiently deep to block the pharynx will cause asphyxia. Initially the airway may be patent through the nose; collections of saliva, excessive mucus with oedema of the pharynx and nasal mucosa, progressively cause complete obstruction. It is almost always homicidal and the victim is usually an infant. Sudden death due to reflex vagal inhibition may occur. **The gag should be examined for buccal epithelial cells.**

Gagging is usually resorted to prevent the victim from shouting for help, and death is usually not intended. The victim's hands are tied behind to prevent their removing the gag, and the legs are tied together to prevent walking or running for help.

OVERLAYING: Overlaying or compression suffocation results due to compression of the chest, so as to prevent breathing. It occurs when the mother or other person shares a bed with an infant. During sleep, the older person rolls on to, or crushes the infant. The thoracic movements are limited and respiratory exchange is either reduced or completely prevented. **In many such cases, the mother or older person goes to the bed under the influence of alcohol.** It is very rare. Flattening of the nose and face may or may not be seen. These parts are pale. The nostrils are often filled with froth, which may be bloodstained and this may stain the pillow or garment. The usual findings are those of asphyxia.

CHOKING

Choking is a form of asphyxia caused by an obstruction within the air-passages.

The foreign body becomes arrested at, or just below the vocal cords and may produce an inflammatory reaction with oedema.

Causes: (1) Choking commonly occurs during a meal when food is accidentally inhaled, especially when the victim is laughing or crying. (2) Vomited matter may be inhaled by a person under the influence of drink or of an anaesthetic, during a fit of epilepsy, or while in a state of insensibility from other causes. (3) Infants usually regurgitate clotted milk after a meal, and this may fall into the larynx. (4) Impaction of solid bodies, such as a large bolus of food, seeds, piece of meat, onion, potato, live fish, mud, leaves, cotton or a set of false teeth may cause asphyxia **(Plate 18-106 and 18-107)**. (5) Gauze packs inserted during an operation can be inhaled and cause death. (6) Children often place objects like marbles or coins in their mouths, which may pass into larynx or trachea during a sudden deep inspiration. (7) Objects like rubber balloons may be inhaled by children during play.

Suicidal Choking: It is rare. The victims are usually mental patients or prisoners. For this a foreign body is thrust into the throat.

Homicidal Choking: Choking may occur by stuffing a piece of cloth or paper into the pharynx or larynx. It is very rare and is practicable only when the victim is an infant or suffering from disability or disease.

Accidental Choking: Choking is almost always accidental. Choking from objects being lodged in the throat is commonly seen in the very young, elderly, psychiatric patients, acutely intoxicated, or in the physically weak, particularly where the ability to swallow or masticate is severely impaired.

Cause of Death: (1) Asphyxia. (2) Cardiac inhibition. (3) Laryngeal spasm. (4) Delayed death may result from pneumonia, lung abscess or bronchiectasis.

CAFE CORONARY: This is a condition in which a healthy but grossly intoxicated person (restaurant patron) who begins a meal suddenly turns blue, coughs violently, then collapses and dies. Death appears to be due to a sudden heart attack. The clinical signs of choking are absent, because high blood alcohol content which anaesthetises the gag reflex.

TREATMENT: A blow on the back or on the sternum may cause coughing and expel the foreign body. If this is not successful, the foreign body should be removed from the hypopharynx with the middle and index fingers or with forceps.

Autopsy: The foreign body, a large piece of poorly chewed food (bolus or a piece of meat), which caused the occlusion of air-passages will be found in the mouth, throat, larynx or trachea. When food or vomited matter has been inhaled, particles of food material may be observed embedded in thick mucus in the trachea and bronchi, and particles may be drawn into the bronchioles.

TRAUMATIC ASPHYXIA: Traumatic asphyxia results from respiratory arrest due to mechanical fixation of the chest so that the normal movements of the chest wall are prevented. Usually there is a gross compression of the chest by a powerful force. Fatal cases are only due to accident.

Causes: (1) Multiple deaths are likely to occur when panic results from an outbreak of fire in a theater, or whenever large crowds gather in an enclosed place. Some are crushed by the weight of the crowd, the chest being pressed violently, or may fall on the ground and crushed under feet. (2) Crushing by falls of earth or stone, usually in a coal mine or during tunnelling or in a building collapse. (3) Sometimes, the victim is pressed to the ground by some heavy weight, as by a motor vehicle or other machinery. (4) Occasionally, it results from indirect compression when the body is subjected to force in such a manner that his thighs and the knees are driven against his chest, the so-called "jack-knife" position.

Postmortem Appearances: An intense cyanosis of deep purple-red colour of the head, neck and upper chest, above the level of compression is the prominent feature (Plate 18-108). Below this level, the skin is pale or mildly cyanosed. Areas of pallor seen at the level of the collar of the shirt, folds or creases in the garments, buttons, braces, etc., are another characteristic feature. The face, lips and scalp may be swollen and congested. Fractures of the ribs, and various other bones may occur. If the patient recovers, the purple colour gradually disappears in 10 to 14 days without the colour changes seen in bruises. Internal organs are congested.

Traumatic asphyxia and smothering: When a person is buried in loose earth or sand or in grain silos, he may die due to occlusion of nose and mouth and immobilisation of chest and abdomen by external pressure sufficient to prevent respiratory movement.

BURKING: Burking is a method of homicidal smothering and traumatic asphyxia. William Burke and William Hare, killed 16 persons in Edinburgh during the years 1927 and 1928, and sold their bodies to Dr. Robert Knox for use as specimens in his anatomy classes. A victim usually alone, or away from the family was invited to their house and given alcohol. Then the victim was thrown to the ground and Burke used to kneel or sit on the chest and close the nose and mouth with his hands, and Hare used to pull him round the room by the feet.

POSTURAL (POSITIONAL) ASPHYXIA: It occurs when an individual acquires a sudden body position in which his breathing is impaired, often because of neck twisting with kinking or compression of the trachea and/or elevation of the tongue into the posterior hypopharynx. In addition, the normal venous return to the heart may be impaired. **Body is typically inverted (upside-down), and the weight of the abdominal contents press against the diaphragm and push it up, compressing the thoracic organs which combined with decreased respiratory movements causes cardiorespiratory**

failure and death. Congestion and cyanosis are prominent, with petechial haemorrhages in the face and neck. (1) It occurs when a person falls into a well and wedged between the walls. (2) It may also occur from forcible flexion of the neck on the chest. (3) An intoxicated person may slide out of bed so that his head and adjoining region hang down from the edge, and the remaining body rests at an upper level, or when he collapses in a narrow space and neck is bent or twisted. (4) Occasionally, it results from indirect compression, when the body is subjected to force in such a manner that his thighs and knees are driven against his chest, the so-called "jack-knife" position. **It is always accidental. There is usually marked congestion, cyanosis, and petechiae.**

DROWNING

Drowning is a type of asphyxia due to aspiration of fluid into air-passages, caused by submersion in water or other fluid. Complete submersion is not necessary, for submersion of the nose and mouth alone for a sufficient period can cause death from drowning.

Duration of Submersion in Fatal Cases: When a person falls into water, he sinks partly due to the force of the fall, and partly to the specific gravity of the body which is 1.08. The specific gravity of fat is 0.92. Shortly afterwards, he rises to the surface due to the natural buoyancy of the body. In sudden immersion into cold water, the victim may take a deep inhalation of water due to reflex from stimulation of the skin. He may hold his breath for varying periods until the CO_2 in his blood and tissues reaches sufficient levels to stimulate the respiratory centre. At that time an inevitable inhalation of water may occur. When he cries for help and struggles, he is likely to inhale water, which produces coughing and drives out large volume of air out of lungs and leads to disturbance of the rhythm of the breathing. He may vomit and aspirate some gastric contents. His struggle increases and again he sinks. If this occurs during inspiration he will inhale more water. The struggle for life with rising and sinking of the body goes on for a variable period, depending on the vitality of a person, until he remains submerged. Convulsive movements then occur, followed by coma or suspended animation and death.

Types: Drowning is of four types: **(1) Wet drowning (typical drowning):** In this **water is inhaled into the lungs**, and the victim has severe chest pain. This is also known as **primary drowning,** in which death occurs within minutes of submersion secondary to cardiac arrest or ventricular fibrillation. **(2) Dry drowning: In this type, water does not enter the lungs, but death results from immediate sustained laryngeal spasm, due to inrush of water into the nasopharynx or larynx.** This is seen in 10 to 20% cases of immersion especially in children and adults under the influence of alcohol or sedative hypnotics. **(3) Secondary drowning** (post-immersion syndrome; **near-drowning**): **Near drowning refers to a submersion victim who is resuscitated and survives for 24 hours**. The person may or may not be conscious. **These persons may develop hypoxaemia resulting in brain damage.** Death may occur from half to several hours after resuscitation in about 20% of cases due to complications, such as pulmonary oedema, metabolic acidosis, chemical pneumonitis, sepsis, cerebral oedema, cardiac arrhythmias, myocardial anoxia. **(4) Immersion syndrome** (hydrocution or submersion inhibition): **Death results from cardiac arrest due to vagal inhibition** as a result of: (a) cold water stimulating the nerve endings of the surface of the body, (b) water striking the epigastrium, (c) cold water entering ear drums, nasal passages, or mucosa of the pharynx and larynx. This is seen in one to two percent of cases of drowning. **Deprivation of oxygen is a factor in all types of drowning.**

The Pathophysiology of Drowning: The pulmonary alveolar lining is semi-permeable. If water enters the alveoli, an exchange of water takes place through the alveolar lining. The extent and direction of this exchange depends on the difference between the osmotic pressure of the blood and the water.

(1) Drowning in Fresh Water or Brackish Water: In drowning in fresh water (0.05% NaCl), two-and-half litres or more of water may be inhaled and absorbed in three minutes. Blood volume may increase by 50% causing a great strain on the heart. Haemodilution leads to haemolysis, haemoglobinemia and haemoglobinuria, marked hyponatraemia and hyperkalaemia. Fresh water alters or denatures the protective surfactant which lines the alveolar wall, while sea water dilutes or washes it away. The increased load causes rapid overburdening of the heart and produces pulmonary oedema, with transudation of protein-rich fluid into the alveolar spaces and hypoxia and secondary metabolic acidosis. Acute respiratory distress syndrome (ARDS) may result from aspiration if the person survives. When water is inhaled, debris plugging the patient's airway and release of inflammatory mediators cause increased peripheral airway resistance with pulmonary hypertension, decreased lung compliance and fall of ventilation perfusion ratios. The oedema fluid contains serum proteins. **The heart is subjected**

to hypoxia, overfilling, sodium deficit and potassium excess. Cardiac arrhythmias leading to ventricular tachycardia and fibrillation occur, probably due to hypoxia and haemodilution. In addition, **hyperkalaemia, may directly damage the myocardium, decreasing cardiac output.** Metabolic acidosis may impair cardiac function.

(2) Drowning in Sea Water: Due to the high salinity of sea water **(usually over 3% NaCl), water is drawn from the blood into the lung tissue, and produces severe pulmonary oedema, and hypernatraemia.** This causes haemoconcentration. Simultaneously, in an attempt to re-establish osmotic balance, salts from the water in the lungs pass into the bloodstream. **Slow death occurs from asphyxia.**

Causes of Death: (1) **Asphyxia:** Inhalation of fluid causes obstruction to the air-passages. Circulatory and respiratory failure occurs simultaneously, due to anoxia of both the myocardium and the respiratory centre. (2) **Ventricular fibrillation:** In fresh water drowning, arrhythmias of the heart beat, ventricular tachycardia and fibrillation cause death within three to five minutes. Severe hyperkalaemia is only a relatively minor factor. (3) **Laryngeal spasm** may result from inrush of water into the nasopharynx or larynx. (4) **Vagal inhibition** is due to icy cold water, high emotion or excitement and unexpected immersion. (5) **Exhaustion.** (6) **Injuries** due to the head striking forcibly against some solid object.

Fatal Period: Death usually occurs in four to five minutes of complete submersion in fresh water and 8 to 10 minutes in sea water.

Postmortem Appearances: External: The postmortem signs are variable. If the body is removed from the water shortly after death, the skin is wet, cold, moist, and pale. Mud, silt, sand, sea-weed, water-weed, algae, etc., may be present on or in the body, such as mouth, nostrils, ears, etc. The **postmortem hypostasis is light-pink in colour** (simulating the colour in CO poisoning), due to oxygenation, but in some cases it is dusky and cyanotic, or it may be a mixture of the two. **Postmortem staining is usually found on the face, the upper part of the chest, hands, lower arms, feet and the calves, as the body usually floats face down, buttocks up, with legs and arms hanging down in front of the body.** The face may or may not be cyanotic, the conjunctivae are sometimes congested. The pupils are dilated. The tongue may be swollen and protruded. Petechial haemorrhages are rarely seen in the skin. Rigor mortis appears early.

Froth: A fine white lathery froth or foam is seen at the mouth and nostrils, which is one of the most characteristic external sign of drowning. Vigorous agitation of the seromucoid secretion and the surfactant with aspirated water and retained air by the violent respiratory efforts converts the mixture of endogenous secretions and drowning medium into froth. **The froth consists of protein and water and the fine bubbles do not readily collapse when touched with the point of a knife.** Froth is usually not stained with blood. If wiped away, it gradually reappears. **Froth is more common in drowning in sea water.**

Cutis anserina (goose-skin; goose-flesh), in which the skin has granular and puckered appearance may be seen, which is most prominent on the thighs. **It is produced by the spasm of the erector pilae muscles** attached to each hair follicle, and can occur in the living when the skin comes in contact with cold water. It may occur on submersion of the body in cold water immediately after death, while the muscles are still warm and irritable. **It is also produced by rigor mortis of the erector muscles.** Agonal contraction of erector pilae is common. It is rarely seen in India, as the water is usually warm. Retraction of the scrotum and penis is due to the same cause and has the same value. These changes have been designated **"reaction phenomenon"**.

Cadaveric spasm: Weeds, grass, sticks, twigs, leaves, gravel, etc., floating in water may be firmly grasped in the hands due to cadaveric spasm. **This strongly suggests that the person was alive when he drowned**, because it indicates the struggle of the person for his life. This is seen rarely. Damaged nails and abraded fingers showing sand, mud, or other materials under the nails due to struggle has the same significance.

Washerwoman's hands and feet: Soddening of the skin occurs due to absorption of water into its outer layer. It is first seen on the fingertips in 2 to 4 hours, and spreads to the palm and the backs of the fingers, and the back of the hand, in that order in about 24 hours. **Wrinkling of the skin begins to appear shortly after immersion, bleaching of epidermis in 4 to 8 hours, and the bleached, wrinkled and**

sodden appearance is seen in 24 hours. Areas protected by clothing develop these changes later. **The skin becomes sodden, thickened, wrinkled and white in colour known as, 'washer-woman's hands'.** Similar changes are seen on the feet (Plate 19-109 and 19-110).

Internal: Lungs: The lungs are voluminous, may completely cover the pericardial sac, and bulge out of chest when the sternum is removed. This is known as **ballooning**, and is due to presence of fluid and air in the bronchi. The impression of ribs is often seen on the lungs. **On section, an oedematous condition due to the presence of watery, frothy, sometimes bloodstained fluid is seen. This has been described as emphysema aquosum.** It is present in about 80% of cases, and is presumptive evidence of death from drowning. **If the victim is unconscious at the time of drowning, mere flooding of the lungs with water, but without formation of froth occurs, which is known as oedema aquosum.** The foam acts as a "check valve" which permits air entry to the lungs, but obstructs air exit from them. **The more powerful inspiratory efforts allow air entry to the lungs, but expiratory efforts are insufficient to expel air, water and foam.** The lungs feel doughy and readily pit on pressure. **The alveolar walls may rupture due to increased pressure during forced expirations, and produce haemorrhages, which when present subpleurally are called "Paltauf's haemorrhages". Paltauf's haemorrhages are shining, pale bluish-red or pink and may be small, or 3 to 5 cm. in diameter.** They are usually present in about 50% of cases in the lower lobes of the lungs, but may be seen on the anterior surfaces of lungs, and the interlobar surfaces. Red and grey patches may be seen on the surface, due to Paltauf's haemorrhages and patchy interstitial emphysema respectively. Usually the lungs are congested moderately, but may be pale due to the forcing out of blood from the lungs. Petechial haemorrhages on the surfaces of the lungs are very rare, or may be absent due to compression of the blood vessels in the interalveolar septa by the water. The **pleurae** may be discoloured by haemorrhages, but **petechial haemorrhages of asphyxial type are rare.**

Lungs in fresh water drowning: In fresh water drowning, the lungs are ballooned but light in weight. They are pale-pink and appear uniformly emphysematous. They retain their normal shape and do not collapse when they are removed from the chest. A crepitus is heard on sectioning and each portion retains its normal shape. On compression, little froth is squeezed out.

Lung in sea water drowning: In salt water drowning, the lungs are ballooned and heavy, weighing up to two kg. They are purplish or bluish in colour, sodden and jelly-like in consistency and pit on pressure. When removed and placed on a flat surface, they tend to flatten out. On sectioning of the lungs, crepitus is not heard. Large amounts of fluid pour out of the cut sections even without compression. The shape of the sectioned portion is not retained. When squeezed, the tissue is found to be filled with fluid in most parts of the lungs, i.e., they are wet and sodden (Table 13-2). **If the body remains in water for several hours, or if the postmortem is delayed for several hours after removal of the body from the water, these changes become less marked, and the difference in appearance between the fresh water and sea water drowning lungs are not clear.**

Table (13-2). Difference in lungs between fresh water and sea water drowning

Trait	Fresh water drowning	Sea water drowning
(1) Size and weight:	Ballooned but light.	Ballooned and heavy; weight up to two kg.
(2) Colour:	Pale-pink.	Purplish or bluish.
(3) Consistency:	Emphysematous.	Sodden and jelly-like.
(4) Shape after removal from the body:	Retained; do not collapse.	Not retained; tend to flatten out.
(5) Sectioning:	Crepitus is heard. Little froth and no fluid.	No crepitus. Large amount of fluid and froth.

Dry lung drowning: In many cases of drowning, relatively dry lungs **(dry-lung drowning)** are observed due to absorption of water from the air-spaces into the pulmonary circulation. This may occur if circulation continues for a short time after removal of the victim from the water or if resuscitative measures are carried out.

Froth: Froth in the air-passages varies from body to body. They may be completely filled by it, but **usually the froth is seen in secondary bronchi and beyond. This is one of the characteristic signs of drowning.** The mucosa of the air-passages is congested. **If there has been delay between death and examination, froth in the lungs and air-passages and overdistension of the lungs is not seen in most cases of drowning.** The fluid in the respiratory passages is of the same nature as the medium in which the body was found, and substances like fine silt, sand, weeds, diatoms, or various forms of algae can be found. If the matter has penetrated deeply into the lung it is useful evidence, but its presence in the trachea may be due to passive entry after death. **If a dead body is thrown into water, due to hydrostatic pressure water passes into the lungs. This "hydrostatic lung" will simulate the drowning lung.** A "drowning lung" may be produced in a body remaining at the depth of two metres for 24 hours. A drowning lung together with frothy fluid is diagnostic. In death due to laryngeal spasm, very little water may enter the lungs, but asphyxial signs are present. In laryngeal spasm, there is no anatomic evidence. It is a diagnosis of inference and exclusion.

Stomach: The stomach contains water in 70% of cases. If the chemical and microscopic nature of the water is same as that of the medium of submersion, it is a valuable confirmatory evidence of drowning. The amount should be measured and examined for foreign substances. **Water is not found in the stomach if the person died from syncope or shock, and in putrefaction.** Occasionally, the individual vomits during the unconscious gasping phase of drowning, and stomach contents may be found in the air-passages.

The small intestine may contain water in about 20% cases. This sign is regarded as positive evidence of death by drowning, as it depends on peristaltic movement which is a vital act. The other organs are congested. Violent respiratory efforts may force some **water into the middle ear** through the Eustachian tube. **Haemorrhages are found in the middle ear** in about half the cases of drowning. **Haemorrhage in temporal bone, or in the mastoid air cells** is seen in large number of cases.

ALTERATIONS IN BLOOD (GETTLER TEST): Normally the chloride content is almost equal in the right and left chambers of the heart and is about 600 mg. per 100 ml. When drowning occurs in fresh water, and the blood gets diluted, the blood in the left side of the heart will show chloride content up to 50% lower than usual. In drowning in sea water, water is absorbed from the pulmonary circulation into the alveolar spaces and the chloride content in the left side of heart shows an increase up to 30 to 40%. A 25% difference in chloride is significant. The test is of doubtful value.

DIATOMS: They are microscopic, unicellular algae. They are usually strongly impregnated with silica and contain chlorophyll and diatomin, a brown pigment. **They resist heat and acid. There are about 25,000 species. They vary considerably in size from two microns to one mm. in length or diameter. Their shape may be circular, triangular, oval, rectangular, linear, crescentic, boat-shaped, etc.** Their presence varies from place to place, and there are seasonal variations at the same place (Fig. 13-13). They occur abundantly in cultivated soils and on surface of moist rocks. Large numbers of free

Fig. (13-13). Diatoms and planktons.

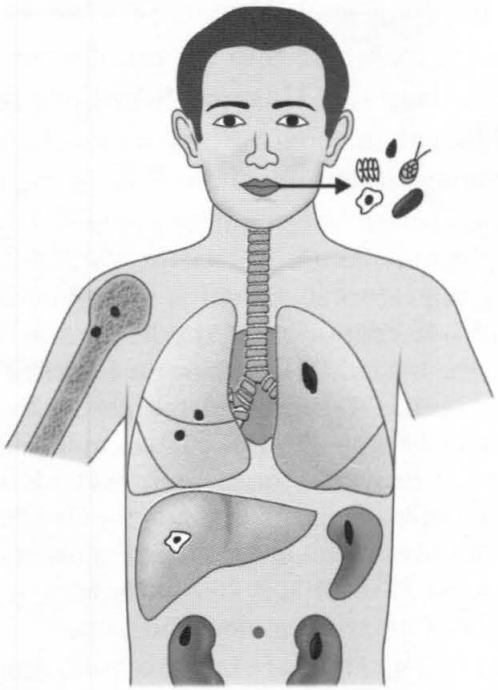


Fig. (13-14). Diatoms in drowning.

floating diatoms are found in both fresh water and sea water. Certain foods, notably shell fish contain large quantities of diatoms. **The drowning fluid and the particles in it, e.g., diatoms and planktons, pass from the ruptured alveolar wall into lymph channels and pulmonary veins and thus enter the left heart.** Only a live body with a circulation could transport diatoms from the lungs to the brain, bone marrow, liver and other viscera, and skeletal muscle (Fig. 13-14). They are also found in the bile and urine. The bone marrow is highly suitable and reliable. The bone marrow of long bones, such as the femur, tibia and humerus or sternum is examined for diatoms. Kidney, lung, liver or brain is also washed and 1 × 1 cm., pieces cut from the deeper tissue can also be examined.

Technique: Five grams of bone marrow or liver, kidney, brain, etc. is put in separate test tubes and covered with five times the volume of concentrated nitric acid, and left at room temperature for one to two days to allow digestion. Alternatively, they can be heated in a water-bath overnight. This process chars, blackens, and destroys organic matter. **Diatoms have silica shells and as such are not destroyed.** The tube is centrifuged, the supernatant acid poured off and replaced with distilled water. This process is repeated two to three times to dilute the acid. The deposit is examined under phase-contrast or dark-ground illumination. **The number of diatoms found in the tissues is relatively small. Strong acid digestion markedly reduces the yield.**

The finding of similar diatoms in the water from the site of drowning and in the body tissues is in favour of drowning. Diatoms from the alimentary canal may enter the circulatory system and reach the various organs in the body, and occasionally may be found in cases other than drowning. **The findings if not absolutely diagnostic are of the greatest value, especially in cases of putrefaction.**

Plasma Specific Gravity: In drowning, the specific gravity of plasma from the left side of the heart is less than that of the plasma in the right side. In non-drowning cases the reverse is the case.

Magnesium: The magnesium content of blood on the left side of the heart is more than 1.25 mg/100 ml. than on the right side in salt water drowning.

Serum strontium: In drowning deaths, serum strontium is raised, which will differentiate death from drowning from postmortem immersion of a body. Differences in the strontium concentration of blood from left and right heart are always greater than 75 µg/l in sea-water drowning.

Diagnosis: The autopsy diagnosis of drowning can pose problems, because the findings are often minimal, obscure, or completely absent. When the findings are negative, the cause of death may be given as "consistent with drowning", or even to admit that the cause of death is "undetermined".

The reliable signs of drowning at autopsy are: (1) Fine, white froth at the mouth and nose. (2) The presence of fine froth in the lungs and air-passages. (3) The voluminous, waterlogged lungs. (4) The presence of water in the stomach and intestines. (5) Finding of diatoms in the tissues. (6) The presence of weeds, sand, mud, etc., firmly grasped in the hands.

The above signs will not be found, if death occurs due to vagal inhibition. In death from syncope, or when a person is in a state of helplessness from drink or other cause, or when a person receives an injury during fall into the water which prevents him from struggle, the signs will be slight. In dry drowning, the postmortem appearances are those of asphyxia. **A body removed from water undergoes rapid decomposition, and if the postmortem is delayed**

for few hours, the signs of drowning will not be found to a great extent.

Decomposition of body: In moderately advanced putrefaction the diagnosis is difficult, the **only evidence being the presence of water in the pleural cavities** due to diffusion of water from the lungs, which finally collapse, and froth in bronchi. In advanced putrefaction, the signs are completely absent.

ACCIDENTAL DROWNING: (1) A sudden collapse from coronary or cerebrovascular disease may cause loss of consciousness, leading to immersion of head in the bath tub and death. (2) Similarly, epilepsy or a fall producing a disabling head injury may also cause death. (3) Other causes include CO poisoning, alcohol or drug intoxication and electrocution. (4) In precipitate labour, the baby may fall into lavatory pan or bucket and die. (5) Occasionally, swimmers, fishermen and dock workers may be drowned accidentally, but it is common in non-swimmers. (6) It also occurs while bathing in tanks, rivers or sea. (7) Women may fall accidentally into a well while drawing water from it. (8) Children may fall in ponds or lakes while playing near their banks.

SUICIDAL DROWNING: In India, drowning is a common method of committing suicide, especially among women and the body is usually fully dressed. If a body is found with heavy weights attached to it, it must be either homicide or suicide and with children homicide alone. The nature of the weights, whether they are tied by ligature or fixed in clothing, or found in the pockets are important. Sometimes, suicides tie their hands or legs together and in such cases the manner of tying and the knots of the rope or ligature should be examined to determine whether they could have been made by the suicide himself. Suicidal drowning may be preceded by the swallowing of the poison, cutting the throat or other suicidal attempts. Injuries may be caused during fall, especially if the bodies are found in the wells.

HOMICIDAL DROWNING: Murder by drowning is very rare, except in the case of infants and children. A person may be pushed into river or into the sea. Homicidal drowning in shallow water is possible, if the assailants hold the victim's head in such a position as to cover the nostrils and mouth. Signs of struggle or marks of violence on the body are likely to be found in such cases.

INJURIES ON DROWNED PERSONS: Wounds may be produced before, at the time of, or after immersion. Before immersion, they may be of accidental, suicidal or homicidal origin. At the time of immersion, they may be produced by the deceased striking hard objects, such as rocks or stone. Immersion of body in water for several hours may cause leaching out of blood from antemortem wounds. There may be no bleeding around these injuries, and as such can be mistaken for postmortem injuries. After immersion, injuries may be produced from striking of the body against rocks, coral or marine structures. Aquatic life (fish, crabs, lobsters, eels, crustaceans, etc.) attack and destroy soft parts of the face, i.e., eyelids, lips, nose, ears, scrotum, penis, and anus. The lesions are circular or oval and punched-out.

Probable Duration of Submersion: The rate of decomposition in an immersed body is reduced to half and doubles when it is removed from water and exposed to air. Very obese persons and infants float more readily than a thin person. The epidermis and nails are loosened, and the skin of the hands and feet may be peeled off like glove or stocking in two to four days. The body floats in about 18 to 24 hours in summer, and 24 to 48 hours in winter in India. In salt water, bodies float earlier than in fresh water. In cold countries the body floats in about two days to one week or more, depending on the temperature of the water. The body usually floats with the spine upper most, though obese persons and some women may float face up due to fat and gas in the breasts and abdomen. In advanced decomposition, the body usually floats belly up.

SEXUAL ASPHYXIAS

(Autoerotic asphyxia or asphyxiophilia)

Partial asphyxia causes cerebral disturbances with feeling of sexual gratification. Pressure on carotid vessels or partial obstruction of air-passages impairs consciousness and may lead to hallucinations of an erotic nature. The degree of asphyxia produced by mechanical means is controlled, but in some cases death occurs accidentally. **These cases are associated with some form of abnormal sexual behaviour, usually masochism and transvestism.** The victims are always males, and usually young. The scene is usually the victim's own house. Adult male homosexuals tend to carry out the procedure in pairs, to prevent accidental death.

Methods: (1) **Hanging is the most frequent form.** The neck is protected by a padding between the

neck and the ligature. The ligature is passed around the neck in the form of a running noose, the free end of which is tied to a limb, or to a fixed object. The noose can be tightened by extending the arms or legs, and when consciousness is lost, the relaxation of the limbs release the pressure on the neck. Such persons are usually found naked, partly naked, or may be wearing women's dress. There may be padding of the brassiere to simulate breasts; female undergarments, and even sanitary pads, wigs and make-up may be worn. Frequently they tie their arms, legs and sometimes waist and genitalia (bondage) with a rope, string, chain, etc. Erotic or pornographic literature and attractive, tempting, female nude photographs are spread out within view. The person may blindfold himself, or may arrange a mirror to watch the events, or camera to make a photographic record. (2) **Sexual gratification may be obtained by electrical stimulation.** For this, electrodes are applied to the genitals or on abdominal wall, usually with a low voltage supply from a battery or transformer. (3) **The head is covered with a plastic or some impervious bag**, which may be secured around the neck by an elastic band. It is sometimes combined with the inhalation of "glue-sniffing". (4) **Inhalation of stupefying substances,** e.g., carbon tetrachloride, trichloroethylene or general anaesthetics may be employed to obtain sexual gratification.

CHAPTER

14 Impotence and Sterility

Impotence is the inability of a person to perform sexual intercourse. Sterility is the inability of the male to beget children, and in the female, the inability to conceive children. A person can be sterile without being impotent, or he can be impotent without being sterile, or both may co-exist. **Frigidity is the inability to start or maintain the sexual arousal pattern in the female.** Frigidity is usually psychological. It can develop in a female who does not experience orgasm in sexual intercourse. Dislike of partner, neurological disorder, hypothyroidism, use of sedatives or depressants, menopause, etc. can cause frigidity. **Sexual dysfunction** is an impairment either in the desire for sexual gratification or in the ability to achieve it.

Legal issues: The question of impotence and sterility may arise in: (A) **Civil:** (1) nullity of marriage, (2) divorce, (3) adultery, (4) disputed paternity and legitimacy, (5) suits of adoption. (B) **Criminal:** (1) adultery, (2) rape, and (3) unnatural offences, where impotency is pleaded as a defence.

Examination: The examination should be undertaken only when asked by the Court or the police. A complete history of the previous illness, especially with reference to nervous and mental condition, and his sexual history should be obtained. A complete medical examination, including central nervous system should be carried out. The condition of the testes, epididymis, cord and penis should be noted and the private parts tested for sensation. Pulsation in the peripheral arteries should be tested. Blood pressure and pulse rate should be measured. In cases of sterility in the male, examination of the seminal fluid is essential. Pressure should be made on the seminal vesicles, and matter expressed is brought to the meatus by pressing the finger along the urethra and examined for spermatozoa.

Opinion: An opinion regarding potency depends upon a man being like or unlike other men. An opinion of diminished potency or impotency cannot be given, unless there is marked deviation from normal. **If the male external genitals are normal, it cannot be said that the person is impotent. In such cases, the opinion should be given in the negative form, stating that from the examination of the male, he finds nothing to suggest that the person is incapable of sexual intercourse.** In the case of the female, the defect usually of the vagina is likely to be clearly seen.

THE CAUSES OF IMPOTENCE AND STERILITY IN THE MALE

(1) Age: The power of erection and therefore of coitus, may be present at a much earlier age than puberty. **Poor physical development of the sex organs is a common cause of impotence, and therefore the medical examiner should depend more on the development of the private parts of the person than on his age.** In cases of precocious development, as in gonadal or adrenal tumours, the sexual organs may show advance development as compared to the body as a whole. In advanced age, the power of erection and the ability to perform the coitus may diminish or disappear but there is no specific age at which such loss of power occurs. Spermatozoa are not usually found before the age of puberty, but may be found in the semen of very old men.

(2) Defects of Development and Acquired Abnormalities: Absence of penis excludes coitus and non-development of penis may prevent the sexual act. Double penis and the penis adherent to the scrotum may cause difficulty in sexual intercourse. In case of partial amputation, sexual act may not be possible. Certain malformations of external genitals, e.g., intersexuality, hypospadiasis and epispadiasis may prevent intercourse, and even when intercourse is possible, the seminal fluid may not reach the vagina, because of the abnormal position of the urethral orifice. Loss of both testicles causes complete sterility

after some time, but may not produce impotence. **If the testes are removed before puberty, impotence is certain, but if the testes are lost after puberty, potency is retained.** The loss of one testis does not affect either potency or fertility. Cryptorchids are not necessarily either sterile or impotent, but sterility is common. With both testes present a man may be sterile due to azoospermia or aspermia.

(3) Local Diseases: Large hernias, large hydroceles, elephantiasis, phimosis, paraphimosis and adherent prepuce may cause temporary impotence by mechanical obstruction to sexual intercourse. Temporary impotency may be caused by acute disease of the penis, such as gonorrhoea, sores on the glans, etc. Diseases of the testicles, epididymis or penis, such as cancer, sarcoma, tuberculosis, syphilis, trauma, etc., may cause sterility, impotence or both. **Fracture pelvis with injury to parasympathetics, fracture spine at L-4–5 level with injury to sacral segments of spinal cord can result in impotence. Bilateral lumbar sympathectomy causes impotence.** Tumours or injury of cauda equina, and spina bifida produce impotence. Lithotomy operation may damage ejaculatory ducts and produce sterility. Exposure to X-rays causes temporary azoospermia. When spermatic cords are blocked due to operation or disease, ligated or cut sterility results.

(4) General Diseases: **Impotence is common during the course of any acute illness;** in convalescence, normal function is rapidly regained. General diseases causing anaemia and debility, e.g., diabetes, pulmonary tuberculosis, chronic nephritis, etc., may cause temporary impotence. Endocrine diseases may produce sexual infantilism and impotence. Certain conditions of the central nervous system, such as hemiplegia, paraplegia, locomotor ataxia, disseminated sclerosis, and syringomyelia may cause impotence, but this is not always so. Paranoia, tabes dorsalis and general paralysis of the insane cause lack of sexual power. Occasionally, the reverse effect, i.e., satyriasis or excessive sexual desire is seen. Temporary impotence is found in neurasthenia. Impotence may also be produced by excessive masturbation. Alcoholism, anabolic steroids, heroin and cannabis can cause erectile dysfunction. Heavy smoking causes impotence due to thrombosis in the penile arteries. Antihypertensives, opiates, psychotropics and tranquilisers, etc. affect the neurotransmitters at the nerve endings resulting in impotence. Orchitis following mumps, especially in adolescence may cause atrophy of testes and may occasionally lead to sterility rather than impotence. Occupational exposure to lead may lead to sterility.

(5) Psychic Causes: **Emotional disturbances are a common cause of temporary impotence.** Fear of impotence or fear of inability to complete the act (first night impotence of the bridegroom) are common causes of temporary impotence, but usually they are soon overcome. Disgust of the sexual act or dislike of the partner may cause temporary or permanent impotence. Quoad hoc (as far as this) is **an individual who may be impotent with one particular woman but not with others.**

Causes of erectile failure: It was thought that psychological factors accounted for most cases of impotence. However, nocturnal penile tumescence have revealed that a **majority of cases of impotence have organic causes. Vasculogenic impotence is one of the most frequent causes of erectile failure (about 40 to 50%).** Vasculogenic impotence may be due to poor arterial inflow into the penis due to arteriosclerotic narrowing of the arteries supplying penis. Diabetes, hypertension, pelvic and genital injuries can cause reduction in blood flow (arteriogenic impotence), or excessive venous leakage of blood from the penis, which can occur in adult life after years of normal sexual life or due to congenital anomalies (venogenic impotence) or both. Other causes are diabetes mellitus (20%), psychogenic (12%), neurogenic (7%), malignancy, testosterone deficiency, trauma, etc. (20%).

CAUSES OF IMPOTENCE AND STERILITY IN THE FEMALE

(1) Age: **As the woman is the passive agent in the sexual act, the age has no effect on potency.** Sexual desire is not completely lost in old age. A woman is usually fertile from puberty to the menopause.

(2) Defects of Development and Acquired Abnormalities: The vagina is sometimes absent in intersexuals, which makes a female completely and permanently impotent and sterile. Impotence may result from some organic defects of the genitals, e.g., total occlusion of the vagina, adhesion of the labia, and the tough imperforate hymen, which can be cured by

surgery. Vaginal injury or severe infection may lead to stricture, and kraurosis vulva in old women may cause narrowing of the vagina. The conical cervix and absence of uterus, ovaries or fallopian tubes produce sterility but not impotence.

(3) Local Diseases: **Hyperaesthesia of vagina, prolapse of the uterus or bladder and vulval or vaginal tumours produce temporary impotence.** Diseases of the genital organs do not cause impotence but may produce sterility, e.g., gonorrhoea involving the cervix, uterus, ovaries and fallopian tubes. Sterility may result from diseases of the ovaries, obstruction of the fallopian tubes or neck of the uterus, rectovaginal fistula, rupture perineum, disorders of menstruation, leucorrhoea, acid discharges from the vagina, etc.

(4) General Diseases: **As the woman is the passive agent in the sexual act, general diseases do not cause impotence.** Occupational exposure to lead or exposure to X-ray leads to sterility. Drug dependence may also lead to sterility.

(5) Psychic Causes: Psychic factors may lead to impotence as in males. **In males, the impotence is passive leading to non-erection, but in females, it is of an active nature, leading to vaginismus. Vaginismus is a spasmodic contraction of the vagina due to hyperaesthesia. It is a classical example of a psychosomatic illness. Anatomically, it may affect the perineal muscles exclusively** or may be felt as a constriction of the levator ani, right up to the vaginal fornices. There is usually a definite cramp-like spasm of the adductor muscles. **Physiologically, these muscle groups contract spastically. Hysterical hyperaesthesia co-exists with this condition**. In extreme cases it may be present all over the vulva and even over adjacent part of the abdomen and thighs. The hyperaesthesia starts at the vaginal introitus. **The spastic contraction of the vaginal outlet is completely involuntary reflex, caused by imagined, anticipated, or real attempts at vaginal penetration. In a fully developed state, constriction of the vaginal outlet is so severe that penetration by the penis is impossible.** If an attempt is made to examine the hymen by passing a glass rod through the hymenal orifice, sphincter muscles of the vagina contract due to which the rod is tightly grasped, and such severe pain is caused that she may scream. In a severe case, the legs cannot be separated sufficiently for the examination of the vulva and occasionally she may rise in a bow-shape, so that she rests only on her head and heels. **Anxiety, stress, fear of pain, guilt complex, etc., can cause strong contractions of paravaginal muscles at the time of intercourse.** It can occur with equal severity in the woman who has borne children as in the virgin.

Aetiology: (1) Male sexual dysfunction (impairment either in the desire for sexual gratification or in the ability to achieve it). (2) Specific incidents of prior sexual trauma. (3) Secondary to dyspareunia. (4) Stimulus derived from attempted heterosexual function by a woman with prior homosexual practice. (5) Rarely, personal dislike or a general feeling of disgust at the idea of coitus. Psychotherapy is beneficial. **Psychological causes affect fertility adversely. A woman may be sterile or impotent with a particular man but not with another. Frigidity is usually psychological**

STERILISATION

Sterilisation is a procedure to make a male or female person sterile, without any interference with potency.

TYPES: (1) Compulsory: It may be carried out on mental defectives and others from a strictly eugenic point of view or as a punishment for sexual criminals. It is not done in India.

(2) Voluntary: It is performed on married persons with the consent of both the husband and wife. (a) Therapeutic: This is performed to prevent danger to the health or life of the woman due to a future pregnancy. (b) Eugenic: This is sterilisation performed to prevent the conception of children who are likely to be physically or mentally defective. (c) Contraceptive: It is performed to limit the size of the family, i.e., for family planning.

GUIDING PRINCIPLES: To avoid legal complications, the following precautions should be taken: (1) The written consent of both wife and husband should be obtained for contraceptive sterilisation. (2) It is not unlawful if performed on therapeutic or eugenic grounds provided there was true and valid consent. (3) The person should be advised to abstain from sexual intercourse for about three months, or until the seminal examination shows absence of spermatozoa on two successive occasions.

ARTIFICIAL INSEMINATION (A.I.)

The artificial introduction of semen into the vagina, cervix or uterus to produce pregnancy is called artificial insemination.

Types: (1) If the semen of the woman's husband is used, it is known as "artificial insemination homologous" or "artificial insemination husband" (A.I.H.). (2) If the semen of some person other than the husband is used it is known as "artificial insemination donor" (A.I.D.). (3) "Pooled" donor semen consists of donor semen to which semen from the husband has been added (A.I.H.D.).

Biological Aspects: Semen is obtained by masturbation and one ml is deposited by means of a syringe in or near the cervix. **The timing of insemination is important**, as the life span of the spermatozoa in the female reproductive tract is short. The time of maximum fertility coincides with ovulation. Because of the problem of timing, insemination on several successive days in the month increase the chances of pregnancy. The success rate is 30% to 40% pregnancies within three to four months of the start of treatment. The use of frozen semen for A.I.D. is becoming increasingly common.

Indications: (1) When the husband is impotent. (2) When the husband is unable to deposit the semen in vagina due to hypospadiasis, epispadiasis, etc. (3) When the husband is sterile. (4) When there is Rh incompatibility between the husband and wife. (5) When the husband is suffering from hereditary disease.

Precautions: Certain recommendations have been made when a donor is used. They are: (1) Consent of the donor and his wife is essential. (2) The identity of the donor must remain secret. (3) The donor should not know to whom the semen is donated and the result of insemination. (4) The donor must be mentally and physically healthy and should not be suffering from any hereditary or familial disease. (5) The donor must not be a relative of either spouse. He should be potent, his age should not exceed 40, and he should have had children of his own. (6) The race and characteristics of the donor should resemble those of the husband of the woman as closely as possible. (7) The donor should be of the same blood group as that of the husband. (8) There should not be any Rh incompatibility between the donor and recipient. (9) The woman to be inseminated and her husband must give consent in writing that an unknown donor should be used. (10) The couple should be psychologically fit and emotionally stable. (11) A witness must be present, when insemination is done. (12) It is usually wise to use "pooled" semen. When husband's semen is mixed with that of a donor, there is always the technical possibility that the husband may, in fact be the father of the child.

DELHI ARTIFICIAL INSEMINATION (HUMAN) ACT, 1995.: The main purposes of this legislation are: (1) to allow the issueless couples to have a child through A.I. and give it a legal status, (2) to control spread of HIV through A.I., (3) to regulate the donation, sale or supply of human semen/ovum for A.I., (4) to make obligatory on the part of medical practitioners: (a) not to indulge in segregating the XX or XY chromosomes, (b) not to disclose the identity of the donor/recipient, (c) to prohibit to carry on semen bank without registration.

Legal Problems: There is no statutory law in India. A.I.H. is justifiable and unobjectionable, since the child is actually the biologic product of both husband and wife. The following are the legal aspects of A.I.D. as applicable to India.

(1) Adultery: The donor and recipient cannot be held guilty of adultery in India.

(2) Legitimacy: The husband is not the actual father of the child, and as such the child is illegitimate and cannot inherit property.

(3) Nullity of Marriage and Divorce: A.I. is not a ground for nullity of marriage or divorce, because sterility is not a ground for it. However, if A.I. was due to impotence, it is a ground. Consent of husband has no bearing on this. When A.I. was done due to the impotence of the husband, the wife may ask for nullity or divorce, even if a child was born out of A.I. If A.I. is done without the consent of the husband, he can sue his wife for divorce and the doctor for damages.

(4) Natural Birth: If the parents do not declare A.I.; the child remains to be a natural child for practical purposes.

(5) Unmarried Woman or Widow: The woman may have a child from A.I. but that child would be illegitimate.

(6) Incest: There is risk of incest between the children born by A.I. and children of the donor.

Complications: (1) The husband may feel humiliation of his deficiency from the presence of child of someone else, and may develop psychiatric symptoms. (2) If the child is mentally retarded or

physically deformed, the father may develop bitter feelings, for he may be held responsible for this deformity by other persons. (3) A neurosis may develop in the mother based on the fact that the child belongs to her alone. (4) She may also develop an obsession to know the donor, and having a second child from the same donor. (5) The child may suffer mental trauma if he learns his past history.

Conception is possible without penetration of the vagina by the penis due to deposition of semen on the vulva, which leads to "fecundation ab extra", the insemination occurring due to passage of spermatozoa from the external genitalia to the uterus. Rhythmic contraction of genital passages and pelvic floor muscles help in it.

TEST TUBE BABIES: The ovum is removed from the ovary of the woman and is fertilised outside the body (*in vitro*) in a small laboratory dish. At the stage of blastocyst, the embryo is returned to the uterus which gets implanted in the endometrium.

SURROGATE MOTHERHOOD: A surrogate mother is a woman who by contract agrees to bear a child for someone else. It is intended to help a couple, of whom the woman is infertile, but the male has no reproductive deficiency. Artificial insemination with the semen of the barren woman's husband is carried out in a hired woman (womb leasing). Surrogate pregnancy can also be done with donated sperm or donated egg or donated embryo. After surrogate birth, the baby is returned to its biological father and his wife. Another method is to remove a mature healthy ovum from the wife and fertilise it "in vitro" with the husband's semen, and implant the embryo in the womb of a hired woman. According to the guidelines of Indian Council of Medical Research (ICMR), the genetic or biological parent names should only be mentioned in the birth certificate. The legal problems of surrogate motherhood are those of artificial insemination donor.

CLONING: It is the technique of producing genetically identical duplicate of an organism artificially. Clone is an organism that has same genetic information as another organism. A clone is said to be all descendants derived asexually from a single individual.

The procedure is known as somatic cell nuclear transplant. Nucleus is removed from an egg cell and nucleus from a somatic cell is introduced into it. The resultant structure is triggered chemically or electrically to develop into an embryo. This is implanted in a surrogate mother. In India, human cloning is officially banned.

CHAPTER

15 Virginity, Pregnancy and Delivery

VIRGINITY

A **virgin** (*virgo intacta*) **is a female who has not experienced sexual intercourse. Defloration means loss of virginity.**

Legal issues: The question of virginity arises in cases of (1) nullity of marriage, (2) divorce, (3) defamation, and (4) rape.

Legally, **marriage** is a contract between man and woman, which implies physical union by coitus. **Divorce** means, dissolution of previously valid marriage.

Genitals: In a virgin, the **labia majora** are thick, firm, elastic and rounded, and lie in apposition so as to completely close the vaginal orifice. They meet in front in the anterior commissure and in back in the posterior commissure in front of the anus. The labia minora are two thin folds of skin just within the **labia majora**. The labia minora are soft, small, pink and sensitive. The lower portions of labia minora fuse in midline and form a fold called **fourchette**. The depression between fourchette and the vaginal orifice is called **fossa navicularis**. The **clitoris** is small and the vestibule is narrow. The **vagina** is about seven-and-half cm long, shorter on its anterior wall (six cm), and longer on the posterior wall (nine cm). It is collapsed to form a slit crosswise of the body. The vagina is narrow and tight, the mucosa is rugose, reddish in colour, sensitive to touch and its walls are approximated. After frequent sexual intercourse (about 15 to 20 sexual acts), the vaginal rugae become less marked, and the vagina lengthens into the posterior fornix. The rugosity is removed only by first birth, but rarely it is absent even in a virgin. The cervical canal is nearly at right angle to the vagina when bladder and rectum are empty. The posterior commissure and fourchette are intact. Fourchette is usually lacerated during childbirth. The hymen is intact. With repeated injury, the fourchette may develop an irregular thickened scar. A single intercourse does not alter the parts much, except the rupture of the hymen.

Hymen: The hymen is a fold of mucous membrane about one mm thick, situated at the vaginal outlet. The average adult hymen consists of folds of membrane having annular or crescentic shape, the broadest part lying posteriorly. Up to the age of 3 to 4 years the hymen is often fleshy and fimbriated. From then until the onset of puberty it is usually a thin, delicate almost translucent membrane. The normal preadolescent hymen is essentially a two-dimensional structure located 2 to 3 cm inside the vaginal introitus. In children, hymen appears as a tense membrane when the thighs are separated. During puberty, it becomes thickened, enlarges and gradually appears as a series of folds often with fimbriated or wavy edge. **The structure of hymen varies considerably. It may be a thin transparent membrane, or it may be tough and fleshy.** The normal hymen lies between these two extremes. **The hymen may be a rigid, fibrous, unyielding structure or it may consist of elastic tissue and be easily distensible.** Recognisable though not severe haemorrhage occurs when hymen is ruptured.

Types: (1) **Semilunar or crescentic:** The opening is placed anteriorly. (2) **Annular:** Opening is oval and situated near the centre of the membrane. (3) **Infantile:** A small linear opening in the middle. (4) **Cribriform:** Several openings. (5) **Vertical:** The opening is vertical. (6) **Septate:** Two lateral openings occur side by side, separated partially or completely by thin strip of tissue. (7) **Imperforate:** No opening.

Fimbriated hymen: The margin of the hymen is sometimes fimbriated (wavy or undulating or markedly irregular), and shows multiple notches

which may be mistaken for artificial tears. **Natural notches are usually symmetrical, occur anteriorly, do not extend to the vaginal wall, and are covered with mucous membrane. Tears caused by sexual intercourse or by foreign body are usually situated posteriorly at one or both sides, or in the midline, usually extend to the vaginal wall, and are not covered with mucous membrane.**

Causes of Rupture of Hymen: (1) An **accident**, e.g., a fall on a projecting substance or by slipping on the furniture or fence or while playing at seesaw. In these cases, tearing of the perineum occurs and usually injuries on other parts of the body will be seen. Hymen does not rupture by riding, jumping, dancing, etc. (2) **Masturbation**, especially with some large foreign body. Hymen is not injured in most cases, because manipulation is usually limited to the parts anterior to the hymen. Labia minora and clitoris are enlarged in such cases. The vaginal orifice may be dilated and edges of the hymen may show scratches. (3) **Surgical operation** and gynaecological examination. (4) **Foreign body**, e.g., solapith introduced into vagina for making very young girls fit for sexual intercourse (*aptae viris*). (5) **Ulceration** from diphtheria, fungus or other diseases. (6) **Scratching** due to irritation of the parts from lack of cleanliness. (7) Sanitary **tampon** may sometimes rupture the hymen.

Breasts: In a virgin, the breasts are firm, elastic and hemispherical with a small undeveloped nipple, surrounded by an areola which is pink in fair complexioned women, and dark brown in dark women. The breasts become large and flabby by frequent handling and sexual intercourse.

Medicolegal Aspects: **The presence of unruptured hymen is a presumption, but is not an absolute proof of virginity.** The diagnosis of virginity is difficult, and in many cases a physical examination of the genital organs may not be helpful. **With an intact hymen, there are true virgins and false virgins.** The hymen is present always in a virgin in some form or other, but very rarely may be absent congenitally. **The important signs of virginity are:** (1) An intact hymen. (2) A normal condition of the fourchette and posterior commissure. (3) A narrow vagina with rugose walls (**Table 15-1**). These signs taken together may be regarded as evidence of virginity, but taken singly cannot be so regarded. The hymen is usually ruptured at the time of first coitus, and at first only presents a torn appearance. Hymen may not be ruptured even after repeated acts of coitus if it is loose, folded and elastic or thick,

Table (15-1). Difference between virginity and defloration

	Trait	Virginity	Defloration
(1)	Hymen	It is intact, rigid and inelastic; the edges are distinct and regular with a narrow opening hardly allowing a small finger to pass	It may be torn or intact; in the latter case it is loose, elastic, with a wide opening allowing passage of two or more fingers
(2)	Labia majora	They are opposed to each other, fully developed and completely close the vaginal orifice	They are not apposed to each other, not prominent and at the lower end vaginal orifice may be seen
(3)	Labia minora	They are in contact and are covered by labia majora	They are not in contact and are exposed and separated from labia majora
(4)	Fourchette	Intact	Torn or intact
(5)	Fossa navicularis	Intact	Disappears
(6)	Vestibule	Narrow	Wide
(7)	The vagina	It is narrow, the rugae more folded, and the vault more conical	After repeated intercourse it usually grows in length, and the rugae are less obvious

tough and fleshy which permit displacement, change of shape, and stretching without rupture. Cases have been recorded of women having sexual relations, of pregnant women and even prostitutes in whom the hymen was intact. In women who are used to coitus, and in those who have borne children, the hymen is destroyed, and small, round, fleshy projections or tags, known as **carunculae hymenales or myrtiformes** are formed around the hymenal ring.

PREGNANCY

Pregnancy is the condition of having a developing embryo or foetus in the female, when an ovum is fertilised by a spermatozoon. It is most likely to occur between the ages of 14 and 45 years, but has been reported much earlier and later.

Medicolegal importance: The question of pregnancy has to be determined in the following conditions. (1) When a woman pleads pregnancy to avoid attendance in Court as a witness. Pregnancy itself is not an excuse, unless it is so advanced that delivery is likely to occur soon, or when she or the child is likely to suffer risk by such attendance. (2) When a woman sentenced to death, pleads that she is pregnant, to prevent execution. If she is found to be pregnant, the High Court shall commute the sentence to imprisonment for life. (3) When a woman feigns pregnancy soon after death of her husband to claim succession to property. (4) When a woman alleges that she is pregnant in order to secure greater compensation when her husband dies through the negligence of some person. (5) In case of divorce, the woman may claim to be pregnant to receive more money for maintenance. (6) To assess damages in a seduction or breach of promise of marriage case. (7) When a woman blackmails a man and accuses that she is pregnant by him, to compel marriage. (8) In case of libel or slander of an unmarried woman, widow, or wife living apart from her husband that she is pregnant. (9) When pregnancy is alleged to be motive for suicide or murder of unmarried woman or widow. (10) In case of alleged concealment of birth or pregnancy and infanticide.

Consent: The written consent of the woman should be taken after explaining the reason for the examination and its possible consequences.

DIAGNOSIS OF PREGNANCY: (I) PRESUMPTIVE SIGNS: (1) Amenorrhoea: This is the earliest and one of the most important symptom of pregnancy. The periods may be missed for some time in unmarried woman after illicit intercourse simply from fear and nervousness. In married woman, the periods may stop for some time, when there is an intense desire for pregnancy.

(2) CHANGES IN BREASTS: Breast changes are quite characteristic in primigravidas but are of less value in multiparae. After the second month, breasts begin to increase in size and become nodular due to hypertrophy of the mammary alveoli. As they become still larger, the superficial veins are seen more distinct and enlarged, the nipples more deeply pigmented and more erectile, and the areola which is pink in the virgin, gradually becomes dark-brown. Around the nipple, the sebaceous glands become enlarged by the end of second month to form small rounded dark-coloured tubercles (Montgomery's tubercles). Colostrum is secreted usually in the third month. Colostrum is thin yellowish fluid consisting of fat globules and large phagocytic cells filled with droplets of fat. After six months, striae are seen especially in primiparae due to the stretching of the skin.

(3) Morning Sickness: It usually appears about the end of the first month and disappears 6 to 8 weeks later. Nausea and vomiting are usually present in the morning, and disappear in a few hours.

(4) Quickening: From about the 16th to 20th week, the pregnant woman feels slight fluttering movements in her abdomen, which gradually increase in intensity. These are due to movements of the foetus, and their first appearance is known as quickening.

(5) Pigmentation of the Skin: The vulva, abdomen and axillae become darker due to the deposition of pigment and a dark line extends from the pubis to beyond the umbilicus, the so-called linea nigra.

(6) Changes in the Vagina: The mucous membrane of the vagina changes from pink to violet, deepening to blue as a result of venous obstruction, after the fourth week. This is known as Jacquemier's sign or Chadwick's sign. The tissues become softer, the secretion of the mucus is increased and pulsation can be felt at an early period.

(7) Urinary Disturbances: During the early weeks of pregnancy, the enlarging uterus presses on the bladder and produces frequent micturition. This gradually disappears after few months as the uterus rises up into abdomen, and reappears a few weeks before term when the head descends into the pelvis.

(8) Fatigue: Easy fatigue is very frequent.

(9) Sympathetic Disturbances: Salivation, perverted appetite and irritable temper are common.

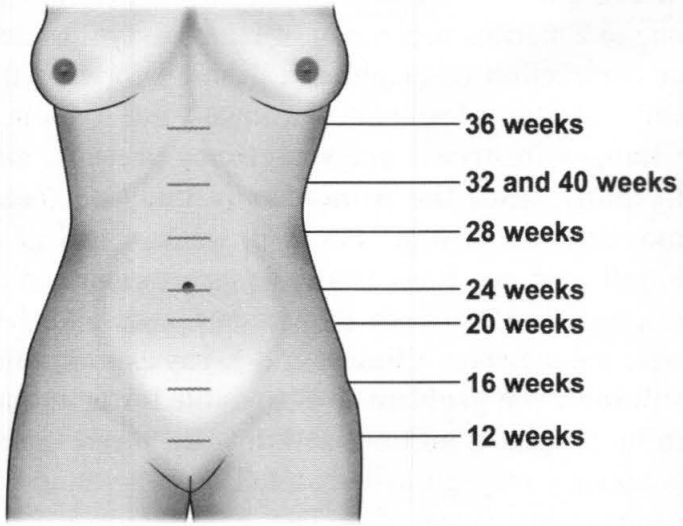


Fig. (15-1). Fundal heights during pregnancy.

(II) PROBABLE SIGNS:

(1) Enlargement of the Abdomen: **By the end of third month the uterus fills the pelvis, and between the third and fourth appears over the brim. At the fifth month it is midway between the symphysis and umbilicus. At the end of sixth month, it is at the level of the umbilicus, and at the seventh month it is midway between the umbilicus and the xiphisternum, and at the end of the eighth month it reaches the xiphoid cartilage (Fig. 15-1). During the last two months, the uterus sinks into pelvis and tends to fall forwards due to its weight. The umbilicus becomes level with the skin by about the seventh month. Striae gravidarum are pinkish or slightly bluish, curved, irregular, depressed lines arranged more or less concentrically, sometimes radially around umbilicus, gradually becoming broader and deeper near inguinal ligament. They also appear on the buttocks and front of thighs. They are seen in late pregnancy (6th or 7th months) and around the time of delivery, become paler with time, and after a year or so appear as white scars (linea albicantes). They are caused by rupture of subcuticular elastic fibres, due to gradual distension of abdomen.**

(2) Uterus: HEGAR'S SIGN **is positive at about the sixth week. If one hand is placed on the abdomen and two fingers of other hand in the vagina, the firm hard cervix is felt, and above it the elastic body of the uterus, while between the two the isthmus is felt as a soft compressible area. This is the most valuable physical sign of early pregnancy.**

(3) Cervix: **From the second month, the cervix progressively softens from below upward which is well marked by fourth month. This is known as** GOODELL'S SIGN. **There is shortening of the cervix towards the last months of pregnancy. The orifice becomes circular instead of being transverse and admits the point of finger to greater depth.**

(4) Intermittent Uterine Contractions (Braxton-Hick's sign): **Intermittent, painless uterine contractions are difficult to be observed before the third month, but are easily felt after the fourth month. Each contraction lasts about one minute and relaxation for about two to three minutes. They are present even when the foetus is dead.**

(5) Ballottement: **It means to toss up like a ball. This is positive during the fourth and fifth months of pregnancy as the foetus is small in relation to the amount of amniotic fluid present. It can be obtained by imparting a sudden motion to the abdominal wall covering the uterus; in a few seconds, the rebound of the foetus can be felt.**

(6) Uterine Souffle: **This is a soft blowing murmur, which is synchronous with the mother's pulse. It is heard by auscultation on either side of the uterus just above inguinal ligament, towards the end of fourth month. It is due to passage of blood through the uterine vessels.**

(7) BIOLOGICAL TESTS: **They are based on the reaction of test animals to chorionic gonadotropins contained in the pregnant woman's blood or urine. They are: (1) The rapid rat test. (2) The Aschheim-Zondek test. (3) Friedman test. (4) Hogben or Female Toad test. (5) Male Frog test. (6) Galli-Mainini test.**

(8) IMMUNOLOGICAL TESTS: **The hormone human chorionic gonadotropin (HCG) and human chorionic somatomammotropin (HCS) are secreted by the syncytial trophoblastic cells into the fluids of the mother. They can be detected in maternal blood on about the eighth day after conception. The rate of secretion rises rapidly to reach a maximum about seven weeks after conception, and decreases to a relatively low volume by sixteen weeks after conception. These tests utilise antibodies to react with another substance for the detection of HCG. Because they are convenient and very reliable, they have replaced bioassays for routine screening. An early morning urine specimen will contain the highest level of HCG and is preferable for testing. Tests are positive 12 to 15 days after implantation.**

(1) Inhibition (Indirect) Latex Slide Test.

(2) Direct Latex Slide Test.

(III) Positive Signs of Pregnancy:

(1) Foetal Parts and Movements: Foetal movements can be felt by placing hands on the uterus from 16 to 18 weeks and may be seen by fifth month. Foetal parts such as head and limbs can be distinctly felt after 24 weeks of gestation.

(2) Foetal Heart Sounds: **They are important and definite sign of pregnancy. They are heard between 18 and 20 weeks for the first time.** The sounds are like the ticking of a watch placed under a pillow. Their rate is usually about 160 at fifth and 120 at the ninth month. They can be monitored and recorded accurately by an electronic foetal monitor. They are not synchronous with the mother's pulse. **Foetal heart sounds are not heard:** (1) When the foetus is dead. (2) When there is excessive quantity of liquor amnii. (3) When abdominal wall is very fat. (4) When examination is made before 18 weeks of pregnancy.

(3) Radiological examination: X-ray Diagnosis: **At about 15 to 16 weeks, foetal parts can be detected with certainty,** but occasionally parts are detected as early as ten weeks. **The shadows to be searched in the pelvis of the mother are:** (1) Crescentic or annular shadows of the skull. (2) A series of small dots in a linear arrangement of the vertebral column. (3) A series of fine curved, parallel lines of the ribs. (4) Linear shadows of the limbs. At a later stage X-ray examination may be of value in the diagnosis of a twin pregnancy, foetal death or abnormality, hydatidiform mole, etc.

Radiological signs of foetal death are: (1) Spalding's sign. (2) Collapse of the spinal column due to absence of muscle tone. (3) Presence of gas in the heart and great vessels.

(4) Ultrasonography: Gestational ring is seen by sixth week, and distinct echoes from the embryo within the gestational ring by seven weeks. Foetal heart beat can be made out by tenth week and, foetal head and thorax by fourteenth week. In 8 to 14 weeks, crown-rump length allows accurate estimation of gestational age. Early identification of dead embryo, multiple pregnancy, vesicular mole, placental site, foetal anomalies, hydramnios and foetal growth is possible. Foetal heart beat can be made out by twelfth week and foetal head and thorax by fourteenth week.

PSEUDOCYESIS (spurious pregnancy): **It is usually observed in patients nearing the menopause, or in younger women who intensely desire children.** Most of the women suffer from some form of psychic or hormonal disorders. Such **patients may present all the subjective symptoms of pregnancy associated with a considerable increase in the size of the abdomen** which may be due to abnormal deposition of fat, or to tympanites, or contraction of diaphragm pushing forward the entire abdominal wall or occasionally due to ascites. **Changes in breast are sometimes present, and in many cases the woman may imagine foetal movements.** In some cases, pregnancy had gone to full term and frank labour pains occurred, which stopped abruptly when the patients were told they were not pregnant. **Clinical and X-ray examination will solve the problem. It is possible for a woman to be pregnant and not to know it.** In rare cases, pregnancy may go as far as full term, without the woman being aware of the fact.

Signs of Pregnancy in the Dead: The presence of an embryo, foetus, placental tissue, membranes or any other product of conception is a positive proof. The uterus is thickened and increased in size. A well-formed corpus luteum is found in one of the ovaries. Even in an exhumed body which has been reduced to a skeleton, foetal bones will be found in the remains.

The Period of Gestation: (1) The Average Period: The usually accepted average is 280 days from the first day of the last menstrual period, so that the actual period of gestation is **about 266 days or less**. In most women, the period of gestation is 40 weeks, or ten times the normal intermenstrual period which is usually 28 days.

(2) The Maximum Period: The law has not fixed limit of gestation. Each case is decided on its own merits. The maximum period according to some authors ranges from 315 to 322 days. As a rule, the longer the gestation, the larger the infant.

(3) The Minimum Period and Viability: **Children born at or after 210 days of uterine life are viable**, i.e., are born alive and able to survive. Children born after 180 days of uterine life may be viable.

POSTHUMOUS CHILD: **It is a child born after the death of its father, the mother being conceived by the said father.** Legal issues involved are legitimacy, inheritance of property, compensation case for slander against the mother.

SUPERFECUNDATION: **It means the fertilisation of two ova which have been discharged from the ovary at the same period, by two separate**

acts of coitus committed at short intervals. The incidence of twin pregnancies is about 1.5% and of these, about 70% are binovular twins resulting from separate fertilisation of two ova formed in the same menstrual cycle. **Development of twins in utero is parallel but not equal, depending on the relative blood supplies from the separately formed placentae.** Both ova do not always develop to maturity. One foetus may be aborted early or die, and retained until the labour that expels the other. **The dead foetus may be flattened by pressure and may not be recognised, and is referred to as foetus compressus or foetus papyraceus.** The spermatozoa causing fertilisation may be from different men. The rare cases, where two ova are fertilised by a white and black person, or persons with entirely different blood groups are the only certain examples of this condition.

SUPERFOETATION: **This means the fertilisation of a second ovum in a woman who is already pregnant. Ovulation may occur during first trimester of pregnancy until decidua vera comes into apposition with decidua reflexa and decidual cavity gets obliterated. Fertilisation of newly released ovum may occur following coitus. Later, two foetuses are born either at the same time showing different stages of development, or two fully developed foetuses are born at different periods varying from one to three months.** Cases where a second fully developed child was born a considerable time after the first have been explained on the assumption of twin pregnancy, in which the second child did not develop either due to diminished blood supply or some other cause. On the birth of the normally developed child, the second foetus received proper nourishment and is born later as mature child. Evidence is accumulating which indicates that superfoetation is not only a possibility but a reality. **Its occurrence in a bipartite or double uterus is certainly possible.**

LEGITIMACY AND PATERNITY

Legitimacy is the legal state of a person born in lawful marriage. If a person is born during the continuance of a legal marriage, or within 280 days after the dissolution of the marriage by divorce or death of the husband, his birth is presumed to be legitimate.

Illegitimacy: The child becomes illegitimate or bastard, if it can be proved that the husband could not possibly be the father of child, by producing evidence that the alleged father is: (1) under the age of puberty, (2) physically incapable to beget children because of illness or congenital or acquired deformities, (3) did not have access to his wife during the time the child was begotten, (4) the blood groups of the child and the alleged father are not compatible.

The question of legitimacy arises in:

(1) Inheritance: A legitimate child only can inherit the property of his father.

AFFILIATION CASES: **A woman may allege a particular man to be the father of her illegitimate child, and file a case in the Court for fixing the paternity (Affiliation cases).** If the paternity is fixed on a certain person, he becomes responsible to support the child. A First Class Magistrate can sanction a monthly allowance of any sum taking into consideration only the necessities of life like food, clothing and lodging, etc. but not luxuries for the maintenance of such child (S. 125, Cr.P.C.). Blood group tests are useful in such cases. DNA fingerprinting will positively fix or exclude paternity.

(2) SUPPOSITITIOUS CHILDREN: **It means fictitious children. A woman may pretend pregnancy and delivery and later produce a child as her own, or she may substitute a male child for a female child born of her, or for an abortion.** This is done for extorting money or for the purpose of claiming property. In such cases, the woman should be examined for signs of pregnancy and delivery. The medical evidence is useful only when the age of the supposititious child does not correspond to the date of the pretended delivery. DNA fingerprinting will be conclusive.

(3) Paternity: The question of paternity arises in contests over legitimacy, posthumous births or supposititious children. It may be determined from: **(a) Parental likeness:** The child may resemble the father in feature and figure and also in gesture and other personal peculiarities. Such evidence even when it is positive is regarded only as corroborative.

ATAVISM: The child does not resemble its parents, but resembles its grandparents. This is due to inheritance of characteristics from remote instead of from immediate ancestors, due to a chance recombination of genes which failed to express in the father, but not in the child. Any mental or physical characteristic or tendency or disease peculiar to a remote ancestor may be inherited. Mutation is a change in a gene between generations resulting in an altered character in the child.

(b) Developmental Defects: Diseases or deformities may sometimes be transmitted from parents to children.

(c) Blood Group Tests or Paternity Tests: Blood groups are transmitted from the parents to the children.

(d) DNA fingerprinting.

DELIVERY

Delivery means the expulsion or extraction of the child at birth. The question of delivery arises in (1) abortion, (2) infanticide, (3) concealment of birth, (4) feigned delivery, (5) legitimacy, (6) nullity of marriage, (7) divorce, (8) chastity, and (9) blackmail.

Signs of Recent Delivery in the Living: (1) **General Indisposition:** For the first two or three days, the woman is pale, exhausted and ill-looking with increase in pulse and slight fever. (2) **Breast changes:** These are full, enlarged and tender with a knotty feeling, and colostrum or milk may be expressed. The areolae are dark, nipples enlarged, superficial veins prominent, and Montgomery's tubercles are present. (3) **The abdomen:** The abdominal walls are pendulous, relaxed, wrinkled, and show striae gravidarum especially in flanks. (4) **The perineum** is sometimes lacerated. (5) The **labia** are tender, swollen and bruised or lacerated. (6) The **vagina** is smooth-walled, relaxed, capacious and may show recent tears which usually heal by the seventh day. The rugae begin to reappear about the third week. (7) The **uterus:** Immediately after delivery, the contracted and retracted body of uterus feels like hard muscular tumour, the upper border of which lies about three cm below the umbilicus. It then diminishes in size by about one-and-half cm a day. On the sixth day, it is midway between the umbilicus and pubis, and returns to normal condition in nine weeks. (8) The **cervix** is soft, canal dilated and its edges torn and lacerated transversely. The internal os begins to close in the first 24 hours. The external os is soft and patent and admits two fingers. At the end of a week, one finger is admitted with difficulty and is closed in two weeks. (9) The **lochia:** It is a discharge from the uterus which lasts for two or three weeks. It has a peculiar sour, disagreeable odour. During the first four to five days, the discharge is bright-red and contains large clots (lochia rubra). During the next 4 days, it becomes serous and paler in colour (lochia serosa). After the ninth day, the colour becomes yellowish-grey or turbid (lochia alba) until its final disappearance. (10) **Intermittent contractions of the uterus** are usually present for the first 4 to 5 days. (11) If blood or urine gives a **positive pregnancy test**, it is strong corroborative evidence that pregnancy has recently terminated. Pregnancy tests are likely to be negative in a week or ten days. The above signs are more characteristic of full term delivery than of a premature one.

Signs of Recent Delivery in the Dead: All the local signs mentioned above may be present. The findings in the internal genitalia will vary with the period of gestation and the time after delivery at which death occurred. Shortly after delivery, the placental site appears as an irregular, nodular, elevated area 15 cm. in diameter, and is covered with clotted blood, lymph and portions of decidua. The placental site measures 3 to 4 cm. in diameter at the end of the second week, and only one to two cm. at the end of six weeks. A large corpus luteum is usually found in one of the ovaries.

SIGNS OF REMOTE DELIVERY IN THE LIVING: A previous pregnancy usually leaves permanent marks on a woman, especially when it has gone to full term. (1) Abdomen: The abdominal walls tend to be lax and show multiple white scars on the lateral aspects. Linea nigra is commonly present. (2) Breasts: The breasts are lax, soft and pendulous, frequently wrinkled if the woman has nursed her baby, and occasionally show linea albicantes. The nipples are enlarged, the areolae dark, and Montgomery's tubercles are usually present. (3) Vulva: The vagina is partially open as the labia do not completely close the orifice. The perineum may show the scar of an old tear. The vaginal rugae are absent and walls are relaxed. (4) Cervix: It is irregular and edges

Table (15-2). Difference in the uterus of parous and nulliparous women.

	Trait	Parous uterus	Nulliparous uterus
(1)	Size:	9 cm. long; 6.5 cm. wide. Larger and thicker.	7 cm. long; 4.5 cm. wide. Smaller and thinner.
(2)	Weight:	80 to 120 g.	40 to 60 g.
(3)	Length:	The body is twice the length of cervix.	The body and the cervix have the same length.
(4)	Fundus:	The top of the fundus is convex and on a higher level than the line of broad ligament.	Upper surface of fundus less convex and on same level as broad ligament.
(5)	Cavity:	The walls are concave from inside forming a wider and rounded cavity.	The walls are convex from inside forming smaller and triangular cavity.
(6)	Cervix:	Irregular, and edges show scars External os is transverse, irregular, and fissured. It may admit tip of the finger; the internal os is not well defined.	Regular, no scars. External os is rounded and appears as a small hollow in the centre of cervix. The mucous membrane is smooth and intact and orifice closed. Internal os circular and well- defined.

show scars. External os is transverse, irregular and fissured, and may admit tip of the finger. The internal os is not well defined. In nulliparous uterus, external os is rounded and appears as a small hollow in the middle of the firm cervix, and internal os is circular and well-defined (Table 15-2).

SIGNS OF REMOTE DELIVERY IN THE DEAD:

The uterus is larger, thicker and heavier. The walls are concave from inside forming a wider and rounded cavity. The body of the uterus is twice the length of the cervix. The cervix is similar to that of remote delivery in the living.

CHAPTER

16 Abortion

Legally, abortion (miscarriage) means the premature expulsion of the foetus from the mother's womb at any time of pregnancy, before the term of pregnancy is completed.

Classification: **(1) Natural:** (a) Spontaneous, (b) Accidental. **(2) Artificial:** (a) Therapeutic, (b) Criminal.

NATURAL ABORTION: Abortion may occur at any time due to natural causes in 10 to 15% of all pregnancies. Abortion is most common about the second or third month. Within the first few weeks, ovum may be passed off without being recognised. In abortions during the first two months of pregnancy, the ovum is expelled intact, covered by the decidua vera. In later months, the foetus is born first, followed by the amniotic sac and placenta, and later on the decidual tissues. The products are expelled, days or weeks after the death of the embryo.

Justifiable or Therapeutic Abortion: **Abortion is justifiable only when it is done in good faith to save the life of the woman, if it is endangered by the continuance of pregnancy.** The World Medical Association adopted a resolution on therapeutic abortion, known as **Declaration of Oslo (1970).**

Criminal Abortion: **A criminal abortion is the induced destruction and expulsion of the foetus from womb of the mother unlawfully,** i.e., when there is no therapeutic indication for the operation. It is resorted to mostly by widows and unmarried women. It is usually carried out before the third month.

Accidental abortion: A general shake-up in advanced pregnancy can produce abortion, but if the ovum is healthy, abortion will not occur.

Unsafe abortion is abortion not provided through approved facilities, and/or persons.

Legal Aspects: Under Sections **312 to 316, of I.P.C.** the punishment for causing criminal abortion may extend from 3 years to life imprisonment.

THE MEDICAL TERMINATION OF PREGNANCY (M.T.P) ACT, 1971

Indications: Under this Act, pregnancy can be terminated under the following conditions. **(1) Therapeutic:** When the continuation of pregnancy endangers the life of woman, or may cause severe injury to her physical or mental health. **(2) Eugenic:** When there is risk of the child being born with serious physical or mental abnormality. This may occur (a) If the pregnant woman in the first three months suffers from (1) German measles, (incidence of congenital defects about 10%). (2) Smallpox or chickenpox. (3) Any severe viral infection. (4) Toxoplasmosis. (b) If the pregnant woman is treated with drugs like thalidomide, cortisone, aminopterin, antimitotic drugs or if she consumes hallucinogens or antidepressants. (c) Mother is treated by X-rays or radioisotopes. (d) Insanity of the parents. (e) Viral hepatitis. **(3) Humanitarian:** When pregnancy has been caused by rape. **(4) Social:** (a) When pregnancy has resulted from the failure of contraceptive methods in case of married woman, which is likely to cause serious injury to her mental health. (b) When social or economic environment, actual or reasonably expected, can injure the mother's health.

Rules: (1) Only a qualified registered medical practitioner having prescribed experience can terminate pregnancy. A registered medical practitioner can qualify if he has assisted in performance of 25 cases of M.T.P. in a recognised hospital. Homoepathy and Ayurvedic graduates are also authorised to conduct MTP. (2) The pregnancy should be terminated in Government hospitals or in the hospitals recognised by the Government for this purpose. (3) Private hospitals may take up abortion if they obtain a licence from the government or district level committee with chief medical officer or district health officer as chairman. (4) The consent of the woman is required before conducting abortion; written

consent of the guardian is required if the woman is a minor or a mentally ill person. Consent of husband is not necessary. (5) Abortion cannot be performed on the request of the husband, if the woman herself is not willing. (6) The woman need not produce proof of her age. The statement of the woman that she is over 18 years of age is accepted. (7) It is enough for the woman to state that she was raped, and it is not necessary that a complaint was lodged with the police. (8) Professional secrecy has to be maintained. (9) If the period of pregnancy is below 12 weeks, it can be terminated on the opinion of a single doctor. (10) If the period of pregnancy is below 20 weeks, two doctors must agree, that there is an indication. (11) Once the opinion is formed, the termination can be done by any one doctor. (12) **In an emergency**, pregnancy can be terminated by a single doctor, even after 20 weeks, even without required training without consulting a second doctor, in a private hospital which is not recognised. (13) The termination of pregnancy by a person who is not registered medical practitioner (person concerned), or in an unrecognised hospital (administrative head) shall be punished with rigorous imprisonment for a term which shall not be less than two years, but which may extend to 7 years.

Confidentiality: The owner/head of the hospital should maintain an admission register for recording details of M.T.P. A number should be assigned to each patient. The name of the patient should not be entered in any case sheet, operation theater register or any other record. The entries in the register shall be made serially for every year. The register is a secret document, which should be kept under safe custody for five years. In all other documents, the number assigned to each patient in the M.T.P. register should be used as a reference number. A certificate can be issued to any employed woman.

M.T.P. (amendment) Bill, 2014, allows homoeopathy and Ayurvedic Practitioners to conduct medical termination of pregnancy.

THE METHODS OF PROCURING CRIMINAL ABORTION

Up to the end of the first month, the woman may take violent exercises, hot baths and purgatives. Up to the end of the second month, when suspicion becomes certainty, abortifacient drugs are used. About the third or fourth month, after failing to procure abortion by the above methods, mechanical interference is done either by the woman herself or by some other person.

(I) Abortifacient Drugs: They either produce congestion of the uterine mucosa and then uterine bleeding, followed by contractions of the uterine muscle and expulsion of the foetus, or they cause the uterine contractions by stimulating the myometrium directly. There is no drug which when taken by the mouth causes abortion without endangering the life of the woman.

(1) Drugs Acting Directly on the Uterus: (A) Ecbolics: They increase uterine contractions and are likely to cause abortion. **Ergot** is most commonly used. It frequently fails during the earlier months of pregnancy. **Quinine** has a direct action upon the uterus or uterine nerves, but its action is not certain. **Lead** in the form of pills made from diachylon (lead oleate) or lead plaster is commonly used. It causes tonic contractions of the uterus and also has a direct toxic effect on the cells of the developing ovum. Death of the foetus may occur in doses which do not seriously affect the mother. Symptoms of lead poisoning may occur before abortion takes place. **Pituitary extract** has a specific oxytocic effect, on uterine muscle. Its effect is significant only near the term. **Synthetic estrogens** do not have any abortifacient effect, except perhaps in very large doses. Decoctions of cotton root bark, nitrobenzol, picrotoxin and strychnine are also used.

(B) Emmenagogues: They produce or increase the menstrual flow. The chief of these are savin, borax, apiol, rue, laburnum, oestrogens, sanguinarin, senecio, caulophyllin, hellebore, etc.

(2) Irritants of the Genito-urinary Tract: They produce reflex uterine contractions, e.g., oil of pennyroyal, oil of tansy, oil of turpentine, cantharides, etc. **Potassium permanganate** is applied to the vaginal vault in 100 to 300 mg. tablets, crystals or solution. It produces acute localised punched-out ulceration with raised edges and a granular black base and causes severe haemorrhage due to erosion of small arterial vessels.

(3) Irritants of the Gastrointestinal Tract: Any substance which causes irritation of the colon may produce hyperaemia and contractions of uterus. Saline cathartics, such as magnesium sulfate or drastic purgatives, such as aloes, calomel, castor oil, croton

oil, jalap, colocynth, phenolphthalein, rhubarb, senna, scammony, podophyllum, elaterium or gamboge are commonly used. The commonly used emetic is tartar emetic.

(4) Drugs having Poisonous Effects on the Body: (A) **Inorganic irritants**, e.g., lead, copper, iron, mercury and antimony, (B) **Organic irritants**, e.g., cantharides, unripe fruit of *papaya,* unripe fruit of pineapple, seeds of carrot, moringa, etc., juice of calotropis, bark of plumbago rosea, caryophyllus, *methi,* saffron, etc.

(II) General Violence: It acts directly on the uterus, or indirectly by producing congestion of pelvic organs, or haemorrhages between uterus and membranes. It may be successful in those in whom there is some natural irritability of the uterus, but in others who are not naturally predisposed to abort, even violence of a severe degree fails to produce abortion.

Intentional: (1) **Severe pressure on the abdomen** by kneading, blows, kicks, jumping, tight lacing, etc., and massage of the uterus through the abdominal wall.

(2) Violent exercise, e.g., horse riding, cycling, jumping from a height, severe jolting as driving over a rough road, running upstairs and downstairs and carrying or lifting heavy weights. Death may occur from rupture of liver, spleen or intestines.

(3) Cupping: A mug is turned mouth downwards over a lighted wick and placed on the hypogastrium and the mug is pulled, which results in partial separation of the placenta. This is usually practised in advanced pregnancy.

(4) Very hot and cold hip baths alternately.

(III) Local Violence: (1) Syringing: The ordinary enema syringe with a hand-bulb is commonly used to inject fluid into the uterus, the hard nozzle being inserted into the cervix. Sometimes, Higginson's syringe is used. The suction valve is placed in a bowl of fluid and pressure applied on the bulb. Due to imperfect filling of the bulb, a mixture of air and fluid is forced into the uterine cavity at a pressure higher than that present in the uterine veins. **The fluid detaches parts of the amniotic sac and placenta from the uterine walls, followed by haemorrhage, uterine contraction, and abortion.** Soap water is often used as an injection material. Irritating substances may be added to the water, such as lysol, cresol, corrosive sublimate, alum, inorganic acids, potassium permanganate, formalin, arsenic compounds, lead compounds, etc. A necrotic pseudo-membrane may form in the vagina and cause severe damage to cervix. Death may result from air embolism. Rough insertion of the syringe into the cervix, or rapid injection of cold or unduly hot fluid may cause sudden death from vagal inhibition. Fear, apprehension and nervous tension act as more potent mechanism for cervical shock. **Douches of hot or cold water may be applied to the vagina or water may be projected with considerable force towards the uterine os.** The injection of fluid can be done by the woman herself or by an abortionist.

(2) Rupturing of the Membranes: The membranes are ruptured by introduction of an instrument, e.g., a uterine sound, catheter, probe, stick, pencil, penholder, umbrella rib, knitting needle, crochet needle, curtain rod, nail, hairpin, piece of wire, glass rod, screw driver, douche cannula, etc., into the cavity of the uterus. **Abortion usually occurs from few hours to two to three days, due to escape of liquar amnii, but occasionally may not occur for days or weeks. Instruments or parts of the instruments can break after introduction into the uterine cavity, or perforation of vaginal or uterine wall.** This can be done by the woman herself, or by an abortionist **(Fig. 16-1)**.

(3) Syringe aspiration: A large syringe attached to a catheter or length of a plastic tubing **can produce suction within the uterus sufficient to rupture the chorionic sac and precipitate abortion.**

(4) Dilation of the Cervix: Foreign bodies left in the cervical canal, such as pessaries, ***laminaria*** **(a dried seaweed) or sea tangle tent (cylinder 5 to 10 cm long) or obturator, dilate the cervix, irritate the uterine mucosa and produce marked congestion and uterine contractions, with expulsion of the foetus.** The cervical canal may be dilated by introducing a **compressed sponge** into the cervix and leaving it there. The sponge swells from moisture in the uterine segment with expulsion of the foetus. **Slippery elm bark** is obtained from a tree grown in America. It occurs in soft flat pieces of varying length and width and about three mm. in thickness. **The pieces are cut to the desired length and breadth and inserted into the cervical canal.** They absorb moisture and within a few minutes a

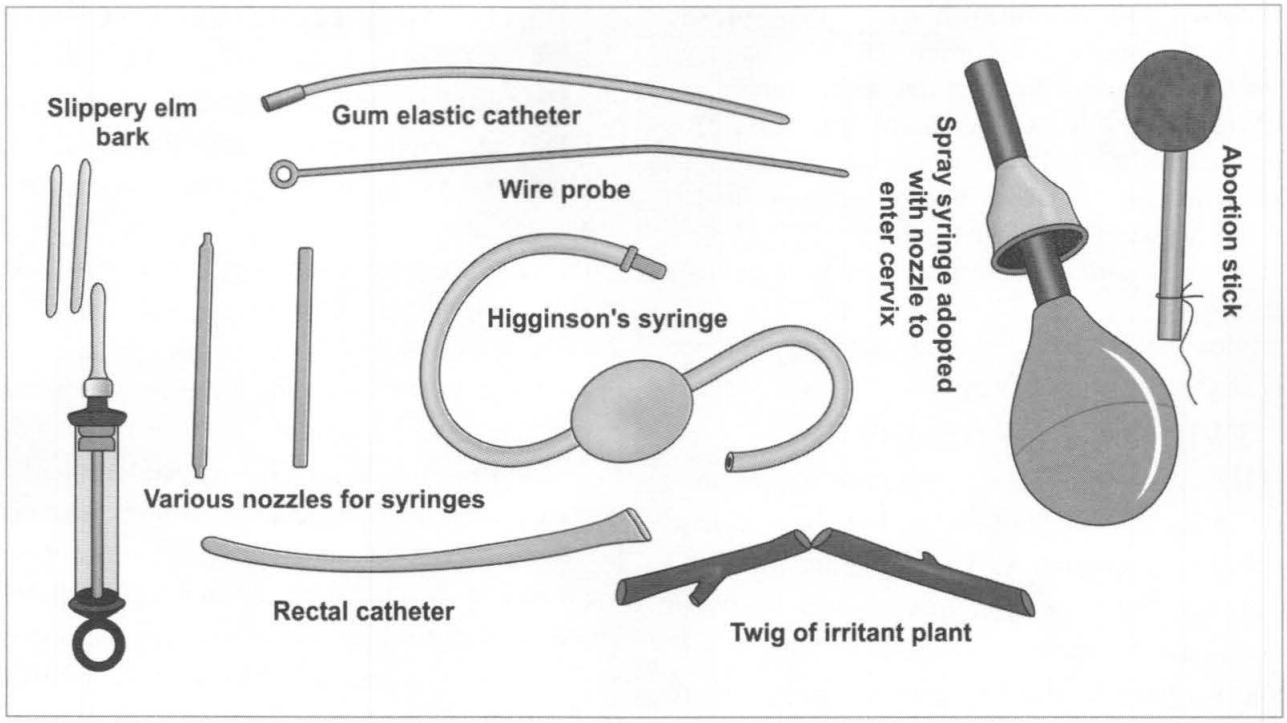


Fig. (16-1). Common instruments used to procure criminal abortion.

jelly-like layer is produced on each side of the bark which is as thick as the bark itself, due to which cervical canal is dilated.

(5) Abortion Stick: This is a thin wood or bamboo stick, from 12 to 18 cm. long. **This stick is wrapped round at one end or for the greater portion of its length with cotton-wool or a piece of cloth, and soaked with juice of marking nut, calotropis, abrus precatorius, asafoetida or paste made of arsenious oxide, arsenic sulphate, red lead, etc. It is introduced into the vagina or os of uterus by professional abortionists *(dhais),* and retained there till uterine contractions begin.** Instead of this stick, a small branch of some irritant plant, e.g., calotropis, nerium odorum, cerbera thevetia, plumbago rosea or zeylanica, etc., is used. In some cases irritating juice is directly applied to the os, or a piece of cloth saturated with irritating juice or paste is introduced into the vagina.

(6) Air Insufflation: Air is introduced into vagina and uterus by various means, e.g., pumps, syringes, and oral-genital contact.

(7) Electricity: The negative pole is placed over the cervix, in the posterior vaginal vault and positive over the sacrum or lumbar vertebrae. When current is passed, uterus contracts and may expel its contents.

(8) Curettage: Some criminal abortions are produced by dilatation and curettage under general anaesthesia.

(9) Pastes: Pastes containing iodine and thymol or mercury (Utus, Foetex, Indulabo, Interruptin) are injected from a collapsible tube with a cannula into the cervical canal. They produce necrosis and infection of the uterine wall and detach parts of the placenta from the uterine wall.

Therapeutic Methods of abortion: (1) Mifepristone (Ru486) inhibits the action of progesterone. This causes breakdown of endometrium and detachment of embryo from the uterine wall. After 36 to 48 hours misoprostol (prostaglandin) is given which produces uterine contractions and the products are expelled within four hours. The procedure resembles a natural abortion. It is not very effective after 7 weeks. (2) **Vacuum Aspiration:** This is done during the first three months of pregnancy. The cervix is dilated and the cannula is introduced into the uterine cavity. Between 8 and 12 weeks of pregnancy, a nine mm. cannula is sufficient. A negative pressure of 0.4 to 0.6 kg/sq.cm is produced in the uterine cavity by means of a vacuum pump for evacuation of the contents. The cannula is moved gently up and down over all sides of the uterine cavity. The contents are broken up by aspiration and are collected in a bottle connected to the cannula. Aspiration usually takes 3 to 5 minutes. The advantages of this method are: it is easy to perform and haemorrhage and danger of perforation of uterus is rare. (3) Low rupture of the membranes. (4) Utus paste injection. (5) Dilation of the cervix and oxytocin infusion, or by a direct injection of ten units of oxytocin into the uterus. (6) In the first three months, dilatation of the cervix and evacuation of the uterus by curettage.

(7) Prostaglandins: Prostin E_2 (PGE_2) and prostin F_2 (PGF_2) induce labour and abortion. They can be given intravenously, orally or by intravaginal application.

(8) Amniotic Fluid Replacement Therapy: This method is useful after 12th week of pregnancy, or in cases of intrauterine death. With long needle, amniotic fluid is removed and replaced with equal volume of 20% saline or 50% glucose. Abortion usually occurs in 24 to 48 hours after injection.

(9) Abdominal Hysterotomy: This method is preferred after fourteen weeks of pregnancy.

EVIDENCE OF ABORTION

IN THE LIVING: The signs of recent abortion: They are those of recent delivery, but they depend on the length of pregnancy. The signs are modified depending on the time that has passed between abortion and examination.

Signs of injury to the abdomen or body should be noted. Any diseases predisposing to natural abortion should be noted. In abortion during the first two to three months of pregnancy, the signs are ill-defined and consist of haemorrhage, slight softening of external os and vaginal walls and slight enlargement of the uterus, which disappear in a few days. The breast changes of pregnancy in the case of primipara are useful. During the fourth and fifth months of pregnancy, the haemorrhage is more marked and the internal os may admit a finger. The os is not injured in abortions of less than six months of pregnancy. The genital organs are much softened and tags of membrane may be found in the uterus. The general condition of the genital tract and the injuries can be made out by visual examination with a speculum. The vaginal canal may show erosions or lacerations. Cervix may show marks of vulsellum forceps, fissures or lacerations, indicating use of an instrument.

Strong antiseptics produce surface tissue necrosis which facilitates bacterial growth. In fatal cases, the sepsis usually involves the endometrium, especially the placental site and of retained products. The myometrium, tubes and adjacent pelvic organs and peritoneum are also infected. Material and liquid from the vagina, uterine cavity and blood should be collected for chemical and bacteriologic examination. Urine, and vomit should be preserved for chemical analysis.

POSTMORTEM EVIDENCE OF CRIMINAL ABORTION

Criminal abortion should be suspected when (1) The deceased is pregnant and deeply cyanosed. (2) Instruments to procure an abortion or abortifacient drugs are found at the scene of death. (3) The underclothing appears to be disturbed after death. (4) Fluid, soapy or bloodstained is coming out of the vagina.

The findings depend upon the mode of abortion practised, and the time which has passed between its performance and death.

Expelled material: The material alleged to have been expelled from the uterus should be examined for products of conception. If recognition is not possible, the material should be placed in water to dissolve the blood. A portion should be examined microscopically, to know whether it is a blood clot, foetus, polyp or fibroid. In the case of blood clot, grouping and precipitin test should be done to know whether it is compatible with the woman's blood groups. In the case of a foetus, the age should be determined **(Fig. 16-2)**.

(1) Abortion by the Drugs: The G.I. must be examined for evidence of irritant poisoning. If such evidence is found, the whole tract and its contents

Fig. (16-2). Criminal abortion (five months pregnancy). The severed foetal head is retained in the uterus

together with other organs of the body should be preserved for analysis. The urinary tract should be examined for signs of inflammation, such as might be produced by cantharides or turpentine. The vagina and cervix should be examined for erosions and inflammation due to local application of irritant and caustic substances, and they should be preserved for analysis.

(2) Abortion by Instruments: (Fig. 16.1) Signs of injury to the abdomen or body may or may not be present. The abdomen is opened, and before removing any parts from the cavity, the peritoneum, pelvic organs and floor are examined for punctures, ruptures, haemorrhage or inflammation. If embolism is suspected, the presence of air in the large veins and in the heart should be looked for, before the organs are removed. The genital organs are removed *en masse.* The uterus is opened first and the presence of foreign bodies, products of conception and the state of uterine wall is noted. Note state of dilatation of cervical canal. Next, the cervical canal is opened and examined for injuries. Wounds of the cervix occur in about fifty percent of the cases, which may be groove-like, parallel notches of the cervical canal, tenaculum marks on the external orifices, cavitation of the wall, fissures, lacerations and perforations. The vaginal canal, and especially the posterior fornix should be examined for signs of lacerations, contusions, perforations and inflammation. The perforations of the vagina and uterus are of different sizes and forms, varying from a small ragged, stellate opening about one centimetre in diameter to much larger tears of stellate, oval or irregular shape. Sometimes, one or more perforations may be present in the fundus. Ragged, stellate or irregular tears of small or large intestine, mesentery, omentum and bladder may occur. Caecum, sigmoid and rectum are usually involved. Segments of the intestine, omentum or mesentery may enter the uterine cavity. The nature of injury may indicate whether the instrument is penetrating and sharp-pointed or blunt-pointed, but the exact nature cannot be made out. Sometimes, foreign bodies may be found in the genital tract. Gross injury when present indicates lack of skill or knowledge of the parts. In skilled hands, instrumentation may leave little or no evidence of any kind, but infection if present is suspicious of interference, unless the foetus is macerated or products are retained. In natural abortion, infection is rare. In self-practised interference the damage is great, but when done by another person the damage is slight **(Table 16-1)**.

Infection: In uterine infection, uterus becomes swollen, spongy and discoloured. The serosal surface may be brownish and endometrium may be ragged, foul-smelling and even purulent **(Plate 20-115)**. Signs of septicaemia may develop with an enlarged, soft spleen, prominent lymph nodes and hepato-renal failure. In extreme cases, kidneys may show bilateral

Table (16-1). Distinction between natural and criminal abortion

Trait	Natural abortion	Criminal abortion
(1) Cause:	**Predisposing diseases.**	**Pregnancy in unmarried woman or widow.**
(2) Marks of violence:	**Not present on the abdomen.**	**May be present on abdomen.**
(3) Genital organs:	**Injuries are not present.**	**Injuries, such as contusions, lacerations, perforations, etc., may be seen in uterus or its contents and vagina.**
(4) Foreign bodies:	**Not present in genital tract.**	**May be present in genital tract.**
(5) Toxic effect of drugs:	**Absent.**	**Erosions and inflammation of vagina and cervix due to local application of irritant and caustic substances may be present. The G.I. or urinary tract may show signs of irritation.**
(6) Infection:	**Rare.**	**Frequent.**
(7) Foetus:	**Wounds absent.**	**Rarely wounds may be present.**

cortical necrosis. In clostridial septicaemia, skin may be bronze coloured, having a mottled appearance (**Table 16-1**).

(3) Abortion by Syringing: There may be fluid in the vagina and soiling of clothes. The mucous plug in the cervix is usually displaced or disintegrated. Corrosion or tissue damage may be seen due to the use of antiseptics. The cervical canal may be dilated and injured. Foamy red fluid may be seen between the uterine wall and foetal membranes with partial detachment of the placenta. The injected fluid enters the uterine sinusoids under pressure, and the fluid and bubbles of gas can be detected in the venous system extending from the sides of the uterus up to right heart. The right side of the heart, the superior and inferior vena cavae, and pulmonary conus contain foamy blood and are "ballooned out". In air embolism, collapse occurs in about two minutes and death in 10 minutes. In self-induced cases, usually, the apparatus is by her side but she may have had time to dispose it off.

Material and fluid from the vagina, uterine cavity, and blood from both ventricles should be collected for chemical and bacteriological examination. Tissues from the uterus, ovaries and other organs must be taken for histological examination.

Fabricated Abortion: Rarely, when a woman is assaulted, she may try to exaggerate the offence by alleging that it caused her to abort. She may acquire a human foetus or an animal foetus to support the charge.

Doctor's Duties in a Case of Criminal Abortion: (1) The doctor should keep all the information obtained by him as a professional secret. (2) He must ask the patient to make a statement about the induction of criminal abortion. If she refuses to make a statement, he should not pursue the matter. (3) He must consult a professional colleague. (4) He must treat her to the best of his ability. (5) If the woman's condition is serious, he must arrange to record the dying declaration. (6) If the woman dies, he should not issue a death certificate, but he should inform the police.

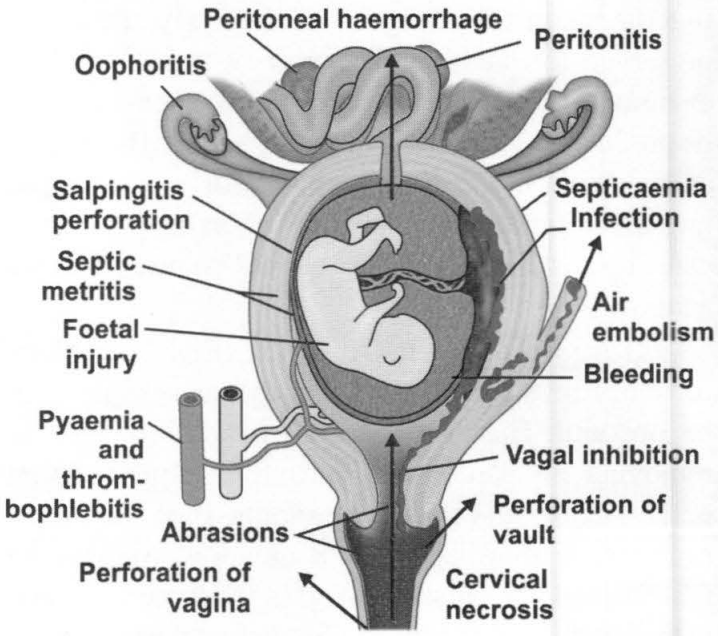


Fig. (16-3). Complications of criminal abortion.

Cause of Death (Figs 16-2 and 16-3): (A) Immediate Deaths: (1) Vagal inhibition. (2) Haemorrhage. (3) Air embolism. (4) Fat embolism.

(B) Delayed Deaths (onset within 48 to 72 hours): (1) Septicaemia. (2) Pyaemia. (3) General peritonitis. (4) Confined local infection and toxaemia. (5) Tetanus.

(C) Remote Deaths: (1) Jaundice and renal failure. (2) Bacterial endocarditis. (3) Pulmonary embolism. Death may result from poisonous effects of drugs.

CHAPTER

17 Sexual Offences

Classification: (I) Natural offences: (1) Rape. (2) Incest. (3) Adultery. **(II) Unnatural offences:** (1) Sodomy. (2) Tribadism. (3) Bestiality. (4) Buccal coitus. **(III) Sexual paraphilias:** (1) Sadism. (2) Masochism. (3) Necrophilia. (4) Fetichism. (5) Transvestism. (6) Exhibitionism. (7) Masturbation. (8) Frotteurism. (9) Undinism, etc. **(IV) Sex-linked offences:** (1) Stalking. (2) Voyeurism. (3) Sexual harassment. (4) Trafficking. (5) Indecent assault.

RAPE

S. 375, I.P.C.: A man is said to commit "rape" if he (a) penetrates his penis, to any extent, into the vagina, mouth, urethra or anus of a woman or makes her to do so with him or any other person; or (b) inserts, to any extent, any object or a part of the body, not being the penis, into the vagina, the urethra or anus of a woman or makes her to do so with him or any other person; or (c) manipulates any part of the body of a woman so as to cause penetration into the vagina, urethra, anus or any part of body of such woman or makes her to do so with him or any other person; or (d) applies his mouth to the vagina, anus, urethra of a woman or makes her to do so with him or any other person, under the circumstances falling under any of the following seven descriptions: (1) Against her will. (2) Without her consent. (3) With her consent, when her consent has been obtained by putting her or any person in whom she is interested, in fear of death or of hurt. (4) With her consent, when the man knows that he is not her husband and that her consent is given because she believes that he is another man to whom she is or believes herself to be lawfully married. (5) With her consent when, at the time of giving such consent, by reason of unsoundness of mind or intoxication or the administration by him personally or through another of any stupefying or unwholesome substance, she is unable to understand the nature and consequences of that to which she gives consent. (6) With or without her consent, when she is under eighteen years of age. (7) When she is unable to communicate consent.

Explanation: (1) For the purposes of this section, **"vagina" shall also include labia majora.** (2) Consent means an unequivocal voluntary agreement when the woman by words, gestures or any form of verbal or non-verbal communication, communicates willingness to participate in the specific sexual act; provided that a woman who does not physically resist to the act of penetration shall not by the reason only of that fact, be regarded as consenting to the sexual activity.

Exception: (1) **A medical procedure or intervention shall not constitute rape.** (2) Sexual intercourse or sexual acts by a man with his own wife, the wife not being under 15 years of age, is not rape.

S. 376, I.P.C.: (1) Whoever, except in the cases provided for in sub-section (2), commits rape, shall be punished with rigorous imprisonment of either description for a term which shall not be less than seven years, but which may extend to imprisonment for life, and shall also be liable to fine. *(2)* Whoever, *(a)* being a police officer, commits rape *(i)* within the limits of the police station to which such police officer is appointed; or *(ii)* in the premises of any station house; or *(iii)* on a woman in such police officer's custody or in the custody of a police officer subordinate to such police officer; or *(b)* being a public servant, commits rape on a woman in such public servant's custody or in the custody of a public servant subordinate to such public servant; or *(c)* being a member of the armed forces deployed in an area by the Central or a State Government commits rape in such area; or *(d)* being on the management or on the staff of a jail, remand home or other place of custody or of a women's or children's institution, commits rape on any inmate of such jail, remand home, place or

institution; or *(e)* being on the management or on the staff of a hospital, commits rape on a woman in that hospital; or *(f)* being a relative, guardian or teacher of, or a person in a position of trust or authority towards the woman, commits rape on such woman; or *(g)* commits rape during communal or sectarian violence; or *(h)* commits rape on a woman knowing her to be pregnant; or *(i)* commits rape on a woman when she is under sixteen years of age; or *(j)* commits rape, on a woman incapable of giving consent; or *(k)* being in a position of control or dominance over a woman, commits rape on such woman; or *(l)* commits rape on a woman suffering from mental or physical disability; or *(m)* while committing rape causes grievous bodily harm or maims or disfigures or endangers the life of a woman; or *(n)* commits rape repeatedly on the same woman, shall be punished with rigorous imprisonment for a term which shall not be less than 10 years, but which may extend to imprisonment for life, which shall mean imprisonment for the remainder of that person's natural life, and shall also be liable to fine.

S. 376A I.P.C.: Whoever, commits an offence punishable under sub-section *(1)* or sub-section *(2)* of section 376 and in the course of such commission inflicts an injury which causes the death of the woman or causes the woman to be in a persistent vegetative state, shall be punished with rigorous imprisonment for a term which shall not be less than 20 years, but which may extend to imprisonment for life, which shall mean imprisonment for the remainder of that person's natural life, or with death.

S. 376B I.P.C.: Whoever has sexual intercourse with his own wife, who is living separately, whether under a decree of separation or otherwise, without her consent, shall be punished with imprisonment of either description for a term which shall not be less than two years but which may extend to seven years, and shall also be liable to fine.

S. 376C I.P.C.: whoever, being (*a*) in a position of authority or in a fiduciary relationship; or (*b*) a public servant; or (*c*) superintendent or manager of a jail, remand home or other place of custody or a women's or children's institution; or (*d*) on the management of a hospital or being on the staff of a hospital, abuses such position or fiduciary relationship to induce or seduce any woman either in his custody or under his charge or present in the premises to have sexual intercourse with him, such sexual intercourse not amounting to the offence of rape, shall be punished with rigorous imprisonment of either description for a term which shall not be less than five years, but which may extend to 10 years, and shall also be liable to fine.

S. 376D I.P.C.: Where a woman is raped by one or more persons constituting a group or acting in furtherance of a common intention, each of those persons shall be deemed to have committed the offence of rape and shall be punished with rigorous imprisonment for a term which shall not be less than 20 years, but which may extend to life which shall mean imprisonment for the remainder of that person's natural life, and with fine.

S. 376E I.P.C.: Whoever has been previously convicted of an offence punishable under section 376 or section 376A or section 376D and is subsequently convicted of an offence punishable under any of the said sections shall be punished with imprisonment for life which shall mean imprisonment for the remainder of that person's natural life, or with death.

S. 228A, I.P.C.: Whoever prints or publishes the name or any matter which may make known the identity of victim of rape (offences under S.376 and 376A to E) shall be punished with imprisonment extending up to two years.

S. 294, I.P.C.: Whoever to the annoyance of others (a) does any obscene act in any public place or (b) sings, recites or utters any obscene song, ballard or words, in or near any public place shall be punished with imprisonment up to 3 months, or with fine or with both.

S. 326A, I.P.C.: Voluntarily causing grievous hurt by use of acid, etc. Imprisonment of not less than 10 years may extend to life and fine of 10 lakh rupees.

S. 326B, I.P.C.: Voluntarily throwing or attempting throw acid (imprisonment of not less than 5 years, may extend to 7 years and fine).

S. 354, I.P.C.: Assault or use of criminal force to woman with intent to outrage her modesty (imprisonment of one year may extend to 5 years and fine).

S. 354A, I.P.C.: SEXUAL HARASSMENT: (1) Physical contact and advances involving sexual overtures, (2) a demand or request for sexual flavours, (3) making sexually coloured remarks, (4) forcibly showing pornography (imprisonment of 1 to 5 years, or with fine or both).

S. 354B, I.P.C.: Assault or use of criminal force to woman or compelling her to be naked with intent to

disrobe in public and even in private if force is used (imprisonment of not less than 3 years, may extend to 7 years and fine).

S. 354C, I.P.C.: VOYEURISM: Imprisonment of not less than one year, may extend to 3 years and fine; for second and subsequent offences imprisonment of 3 to 7 years).

S. 354D, I.P.C.: STALKING: Whoever follows a woman and contacts or attempts to contact such woman to foster personal interaction repeatedly, despite a clear indication of disinterest by such woman, or whoever monitors the use by a woman of the Internet, e-mail or any other form of electronic communication, imprisonment up to 3 years and fine).

S. 370, I.P.C.: TRAFFICKING OF PERSON: Whoever for the purpose of exploitation (a) recruits, (b) transports, (c) harbours, (d) transfers, (e) receives a person, by (1) using threats, (2) using force or coercion, (3) by abduction, (4) by fraud or deception, (5) by abuse of power, (6) by inducement, including giving payments or benefits commits the offence of trafficking (imprisonment for 7 to 10 years).

S. 370A I.P.C.: Whoever knowingly that a minor has been trafficked, engages such minor for sexual exploitation will be punished with imprisonment for not less than 5 years, but may extend to 7 years and fine.

EXPLANATION: Exploitation includes prostitution or other forms of sexual exploitation, slavery or forced removal of organs (consent of the victim is immaterial).

S. 493, I.P.C.: Cohabitation caused by a man deceitfully inducing a belief of lawful marriage is punishable with imprisonment up to 10 years.

S. 498, I.P.C.: Enticing or taking away or detaining with criminal intention a married woman is punishable with imprisonment up to 2 years.

S. 509, I.P.C.: Whoever intending to insult the modesty of any woman, utters any word, makes any sound or gesture, or exhibits any object, intending that such word or sound shall be heard, or that such gesture or object shall be seen by such woman, or intrudes upon the privacy of such woman, shall be punished with simple imprisonment up to 3 years and also fine.

S. 53A, Cr.P.C.: (1) When a person is arrested on a charge of committing an offence of rape or an attempt to commit rape, and there are reasonable grounds to believe that an examination will afford evidence as to the commission of the offence, it shall be lawful for a registered medical practitioner (RMP) employed in a hospital run by the government or by a local authority, and in the absence of such a practitioner within the radius of 16 km from the place where the offence has been committed, by any other R.M.P. acting at the request of a police officer not below the rank of a sub-inspector to examine the arrested person with use of reasonable force as required. (2) The doctor has to examine the person without any delay and prepare a report.

S. 327(2), Cr.P.C: The inquiry into and trial of sexual assault or an offence under S. 376 and 376 A to E of I.P.C., shall be conducted ***in camera*** **(in secret)** and forbids the disclosure of the identity of the victim. **It means the judge and staff along with both parties and their lawyers will be inside the court room.** However, if the judge thinks fit on an application made by either of the parties, may allow a particular person to have access to, or remain in the room used by the court.

S. 375C, Cr.P.C: All hospitals, public or private, shall immediately provide first-aid or medical treatment, free of cost, to the victims of any offence covered under S. 326A (voluntarily causing grievous hurt by use of acid, etc.). S. 376, S. 376A to E (rape) of I.P.C. and shall immediately inform the police of such incident.

Statutory Rape: It is sexual intercourse with a girl below 18 years of age even with her consent. It is neither violent nor physically coerced.

Gang rape or group rape occurs, when a **group of people participate in the rape of a single victim**. Each of the persons shall be deemed to have committed gang rape.

Date Rape: It is sexual intercourse with a woman, who is **given a drink containing a sedative drug like GHB, Rohypnol (flunitrazepam), ketamine, etc., in a party without her knowledge**. These drugs are called rape/club drugs.

Marital Rape: It is forceful sexual intercourse with wife who is living separately from him under a decree of separation, or any custom or usage without her consent.

Consent: A woman of eighteen years and above can give valid consent for sexual intercourse. The consent must be free and voluntary, and given while she is of sound mind and not intoxicated. The consent should be obtained prior to the act. Even a prostitute cannot be forced to have intercourse against her will.

Consent is not valid: (1) When it is obtained by fraud, as by impersonation of the husband or by misrepresentation of facts. (2) When it is obtained by putting her or any person in whom she is interested, in fear of death or hurt. (3) When obtained from a

woman who is of unsound mind, or is in a state of drunkenness. (4) When the woman is below 18 years of age.

Presumption of Consent: The chief evidence of absence of consent is signs of resistance, which is naturally expected from a woman unwilling for a sexual intercourse forced upon her. Such a resistance may cause tearing of clothes, and personal injuries on the body, and even on her private parts. **It is necessary to prove that maximum resistance was offered by the woman, and that all means had been tried to prevent sexual intercourse,** e.g., shouting, crying, beating, biting, etc. Women who faint due to fear, or are made helpless due to their clothes being thrown on their face, or who have been drugged, or made unconscious from any cause, and children may not be able to resist. The resistance offered depends upon the type of woman, her age, development and on the social status.

What Constitutes Rape? The slightest penetration of the penis within the vulva (labia majora), with or without emission of semen or rupture of hymen constitutes rape. Completed act of intercourse is not necessary. There should have been not only an assault but actual penetration. **Rape can be committed even when there is inability to produce erection of penis. Rape can occur without causing any injury, and as such negative evidence does not exclude rape. The doctor should mention only the negative facts, but should not give his opinion that rape has not been committed.** Corroboration by eyewitnesses or circumstantial evidence is necessary in such cases.

Burden of Proof: The prosecution has to prove all the elements of the offence. In many cases of rape, there are no signs of injury, or intoxication by stupefying drugs, and the entire allegation of lack of consent is based on fear and on fraud.

Age of the Accused: In India, there is no age limit under which a boy is considered physically incapable of committing rape. The Court decides the question of his potency from the evidence in the case. Even old men commit rape on young girls.

Age of the Victim: No age is safe from rape, as children of one year or less and old women of 85 years have been raped. Children are more frequently raped than adults as they cannot offer much resistance, and also due to false belief that venereal diseases are cured by sexual intercourse with a virgin.

EXAMINATION OF THE VICTIM

The Union Health Ministry has asked all hospitals to set up a designated room for forensic and medical examination of victims of rape, besides outlawing the two finger test performed on them.

Objects: The objects of medical examination are: (1) To search for physical signs (injuries) that will corroborate the history given by the victim. (2) To search for, collect and preserve all physical (trace) evidence for laboratory examination. (3) To treat the victim for any injuries and against venereal disease or pregnancy, and prevention or lessening of permanent psychological damage.

General Procedure: The following is an outline of a planned procedure. (1) The victim should be examined if requisition is given by the investigating police officer or the Magistrate. The victim of rape may go directly to the doctor, who should examine her and inform the concerned police station, so that the due legal process can be set in motion. The Court or the police have no power of forcing a woman for medical examination against her will. (2) The written, witnessed consent of the woman, and if she is under 12 years, or of unsound mind, the consent of her parents must be obtained in writing. (3) The victim should be identified by the escorting police constable, whose name and number should be recorded. Identification marks should also be noted. (4) The name of the victim and her parent, marital status, residence, occupation, time, date, year, place of examination, and by whom requisition is given should be noted. (5) The victim is examined in the presence of a third person, preferably a female nurse or a female relative of the woman. (6) The examination should be carried out without delay. Swelling and tenderness of vulva may disappear in a few hours. (7) Statements of the victim and of others with her are recorded separately of: (a) preliminary affairs, (b) date, time and place of alleged offence, (c) exact relative positions of the parties, (d) details of struggle or resistance, (e) calls for help, (f) was any pain experienced either at the time of the incident or subsequently, (g) did ejaculation take place during the act, either within the vagina or outside, (h) appearance of any discharge, (i) was there bleeding from vagina, (j) whether consciousness was lost at any time during the incident, (k) details of the events after the alleged assault, (l) the time of the first complaint, and if there was any undue delay, the reason for such a delay. This must be taken down verbatim. The degree of agreement of various

statements will be strong proof of their truth or the contrary. (8) Previous history with regard to sexual experience, menses, vaginal discharge, venereal disease, pregnancies, pelvic operations, etc., should be recorded. (9) The age should be determined and the height and weight recorded. (10) The physical development should be noted in order to determine her capacity for struggle and resistance. (11) Her general behaviour (distressed, dishevelled, dazed or shocked, intoxicated, excited, calm, tearful, aggressive, etc.) and mental state should be observed while she tells her story. (12) The gait should be observed; whether she complains of pain on walking, on micturition or defecation. (13) If it appears that she is under the influence of alcohol or drugs, it must be noted and samples of blood and urine should be collected. (14) An attempt at undressing the woman should not be made, but she should be requested to undress herself. (15) If the victim is in menstrual period, a second examination should be done after stoppage of menstruation.

EXAMINATION PROPER: (1) CLOTHES: Find out whether the clothes are those worn at the time of assault, or changed. Each item of clothing should be examined for stains (blood, seminal, mud, grass, etc.), soiling, tears and loss of buttons, and the site and type of damage. If the offence has been committed in an open place, corroboration can sometimes be obtained by finding grass, leaves, mud, etc., on the buttocks or on the back. In certain cases, stains may be present on pieces of material or handkerchief used by the victim after assault for cleaning purposes. Vulval pads and vaginal tampons should be preserved whether worn at or after the time of the incident. Clothes are very important in corroborating or contradicting her story. Foreign hair, fibres, etc., found on the clothes or on the skin surface must be preserved and compared with those found on the accused.

(2) GENERAL EXAMINATION: The whole body must be examined for marks of violence, especially scratches or bruises resulting from struggle, as regards their appearance, extent, situation and probable age. Marks of violence, may be found: (1) about the mouth and throat, produced while preventing her from calling for help, (2) about wrists and arms where the man held her, (3) on the back from pressure on gravel or hard ground, especially shoulders and buttocks, and (4) about the inner sides of thighs and knees caused by forcing her legs apart, (5) on breasts by rough handling. (6) True bite marks and lovebites are usually found on the breasts, neck, chest wall, and also on the lower abdomen, and upper parts of thighs. The nature and situation of the general injuries should correspond to the victim's description of the assault. The woman usually scratches the assailant during the struggle, and any damaged fingernails should be noted and debris under the nails should be removed and examined for epidermal cells, blood, fibres, etc. **Marks of general violence are likely to be found in one-third cases. Absence of general injuries may be due to:** (1) Submission of the victim due to fear of injury or death, etc. (2) The force used or the resistance offered is insufficient to produce an injury. (3) Bruises may not be noticed for 48 hours following assault. (4) A delay in reporting the incident during which minor injuries will fade or heal.

(3) SPECIAL LOCAL EXAMINATION: The woman is placed on a table in good light, with her legs drawn up and widely open. A vaginal speculum should be used if possible.

Genital injuries are present in one-fifth cases because: (1) The alleged sexual act may consist of only rubbing or touching the genitalia. (2) The victim is sexually experienced. (3) The elasticity of genitalia and hymen in a post-pubertal female. (4) The use of lubricants.

(A) RAPE ON A VIRGIN: Examination of hymen: The labia are separated by gentle traction to examine the hymen. **Rupture of hymen occurs with the first intercourse, which is the main evidence of rape in a virgin.** As the penis enters the genitals, it tends to compress the labia both anteriorly and laterally, producing **bruising of both the labia minora and the labia majora. The amount of bruising will depend upon the force used.** Further penetration forces the penis backwards, because the symphysis pubis prevents anterior movement, and the **hymen is torn posteriorly**. **The character and extent of injury varies in different cases depending upon** (1) the nature of the hymen, (2) disproportion between male and female parts, (3) extent of penetration, and (4) amount of force used. **Tearing usually occurs posteriorly at the sides, between 5 and 7 o' clock position, or in the midline of the hymen. Tears usually occur in the posterior midline of the hymen because the hymen lies suspended across a potential space, whereas anteriorly the periurethral tissues buttress the hymen.** More than one tear may occur. **Several hymenal lacerations indicate first sexual**

intercourse. Any object passing through the hymenal orifice which is larger than its original opening will cause a V-shaped cleft or clefts. **One deep tear may be seen at 6 o' clock position or a number of tears usually in the posterior half of the membrane.** In prepubertal children, the posterior tear of the hymen may involve fourchette, producing a deep U-shaped defect (Fig. 17-1). **Soon after the act, the margins of the torn hymen are sharp and red which bleed on touch, the tissues round about them are swollen and tender. In 24 hours, margins are oedematous, swollen with sero-sanguineous oozing. For 2 to 3 days, there be pain, congestion with oozing of blood-stained fluid.** After three to four days of offence, the edges of laceration are congested and swollen, which completely heal in a week, but they do not unite. With healing, over a period of months V-shaped tears become rounded and appear as U-shaped defects. Rupture of the hymen due to sudden stretching can be caused by agents other than the penis, such as fingers and therefore, evidence of local injury is not proof of penetration. **Frequently, in the absence of hymenal tearing, there is abrasion and bruising of the hymen, and the vaginal orifice** (Fig. 17-1).

In **fimbriated hymen,** a moistened swab can be used to visualise the edges of the hymen. If the hymenal opening is not seen, application of a few drops of warm sterile water or saline on to the hymen will reveal hymenal edges.

Close examination of the hymen: A glass rod (Glaister-Keene rod) with a small spherical head warmed to body temperature, should be passed through the hymenal orifice, spherical head first. Then, it is passed round the posterior surface of hymen, which is slightly stretched by separation of labia minora. In this way, the edges of the hymen become slightly everted. By slowly rotating the sphere around the edges, natural notches are easily differentiated from tears, recent or old. This method does not cause either pain or injury. (2) Another method is to pass a finger into the rectum above the perineal body, and push the posterior vaginal wall forwards and downwards. This pushes the hymen forwards which is clearly seen entirely, lying against the posterior vaginal wall. (3) **Hymenoscope gives excellent transillumination of hymen to detect tears.**

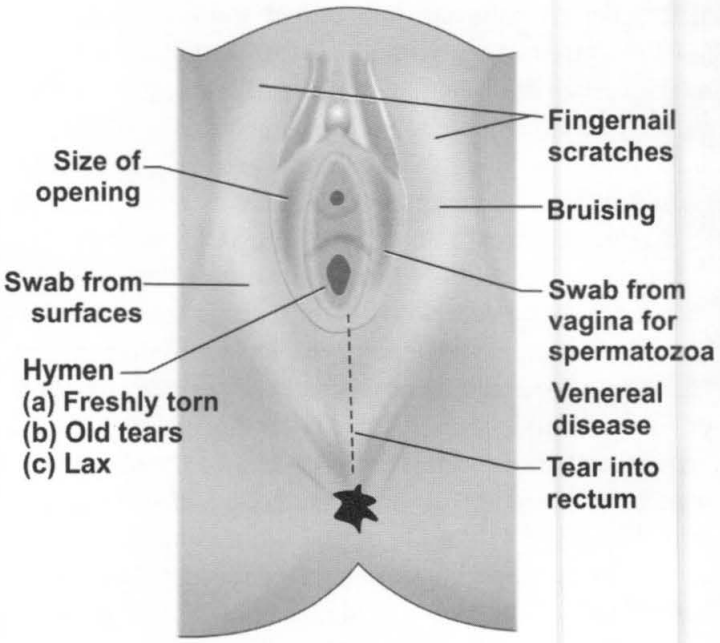


Fig. (17-1). Examination for evidence of rape.

External genitals: Injury to labia is not common, but fingernail scratches may be present on the labia, particularly the labia minora. The labia may be red and inflamed with slight oedema of the vaginal introitus if it is the first sexual act. Redness of the labia minora may be due to uncleanliness. **Swelling and congestion of the mucosa at the introitus, the clitoris and the labia minora are caused by genital stimulation,** but they may also be caused by digital stimulation or masturbation. **These signs usually fade in 1 to 2 hours.** Swelling and tenderness of the labia minora may indicate sexual activity. **The fourchette is fragile and may tear during first intercourse.** Fossa navicularis disappears. If there is disproportion between the male and female genitalia, bruising and lacerations of external genitals may be present with redness, swelling and inflammation. The **posterior commissure is usually intact in the virgin, but may rupture at first intercourse,** especially if there is much disparity in the size of the penis and vagina.

Vagina: Bruising of the vagina is seen as dark-red areas against the overall redness of the vaginal mucosa, and within 24 hours the colour becomes deep-red or purple. It is more frequently seen on the anterior vaginal wall in the lower third, and on the posterior wall in the upper third. Bruising

of this nature is more consistent with penetration of the penis, than with digital penetration. These types of injuries can occur during consenting sexual intercourse. In rape or digital penetration without consent, where preliminary stimulation has not taken place, initial lubrication will be lacking, due to which more severe local bruising or abrasion can result. **Frank laceration of the vaginal wall or vault is rare following sexual intercourse in women of childbearing age,** but it can occur in very young children and in the atrophic post-menopausal vagina. With violent intercourse or where there has been considerable disproportion between the penis and the vagina, laceration of the vaginal wall occurs posteriorly. **The majority of adult rapes are associated with a sudden forcible dilation resulting in some degree of local injury. Bruising, abrasion or laceration are at all times consistent with forcible intercourse with a consenting woman, and do not necessarily indicate rape.**

Abrasions of the cervix and vagina occur almost invariably due to vaginal penetration, and usually due to digital rather than penile penetration.

Vaginal examination: In all cases where there are no fresh injuries, if the state of hymen permits, vaginal examination should be carried out to assess: (1) the laxity of the vaginal orifice, (2) the length of the vagina into the posterior fornix, (3) the number of fingers that can be introduced through the hymenal orifice, (4) elasticity of the hymen, and to determine the degree of penetration which would be possible without its rupture, (5) the areas and the degree of the tenderness complained of by the patient. **In most young women, a finger may be inserted into vagina although the hymen is intact,** which is felt as a constricting ring round the tip of the finger. **If the vaginal opening is enough to admit two fingers easily, the possibility of sexual intercourse having taken place may be inferred.** Two finger test was banned by the Supreme Court of India in 2013, holding that it violated the right of rape survivors to privacy. A speculum may be introduced into the vagina to inspect the interior of the vagina for signs of bruising, abrasion, laceration, etc.

(B) RAPE ON DEFLORATE WOMEN: In deflorate, women even without childbirth, the hymen is completely destroyed, the vaginal orifice dilated and the mucous membrane wrinkled and thickened. **Complete penetration can occur in such women without any evidence except semen. The only proof that penetration has occurred, is presence of spermatozoa in the vagina.** Therefore, the **absence of injuries under certain circumstances does not exclude even complete penetration.** In a married woman, marks of violence to the genitalia are less likely to be found, but they must be looked for, because usually rape is associated with greater violence than sexual intercourse with consent. **In case where there has been resistance, local injury will be present. The vagina may show some deep injury, laceration or bruising with effusion of blood, and swelling and inflammation of the vulva, even when no marks of violence indicating a struggle may be found externally.** Tearing or perforation of the vagina may occur when it is thin or fragile. When older women are raped, senile atrophy and friability of their genitalia result in extensive vaginal lacerations and perineal trauma. In women who have been used to sexual intercourse, injuries from rape usually disappear or become obscure in 3 to 4 days. When there has been much violence, the signs may persist longer. **The presence of violence in other parts of the body is the chief evidence of the crime.**

(C) RAPE ON CHILDREN: In young children, there are few or no signs of general violence, for the child usually has no idea of what is happening, and also incapable of resisting. The hymen is deeply situated, and as the vagina is very small, it is impossible for the penetration of the adult organ to take place. Usually, the penis is placed either within the vulva or between the thighs. As such, the hymen is usually intact, and there may be little redness and tenderness of the vulva.

Injuries: During penetration, the penis compresses the labia both anteriorly and laterally, producing **bruising of both the labia majora and minora.** Further penetration forces the penis backwards, and **the hymen shows a linear tear in the posterior or posterolateral quadrant, which may extend into the posterior vaginal wall and on to the skin of the perineum. Circumferential tears of the mucosa of the vestibule are common. Full penile penetration produces bruising of the vaginal walls with extensive haematoma affecting particularly the**

anterior half, and frequently tears of the anterior and posterior vaginal walls. Anterior tears can involve the bladder and the posterior tears, the anorectal canal. Vaginal vault may rupture and there may be vaginal herniation of abdominal viscera. **The younger the child, the more widespread are the injuries.** In digital penetration of the infant vagina, there is frequently some scratching or bruising of the labia and vestibule, but the circumferential tears are absent. Any attempt to separate the thighs for examination causes great pain, because of the local inflammation. The child walks with difficulty due to pain. Speculum examination should not be performed on the prepubertal child. **The absence of marks of violence on the genitals of the child, when an early examination is made is strong evidence that rape has not been committed.**

(4) CORROBORATIVE SIGNS OF RAPE:

(A) SEMINAL FLUID: The thighs, pubic hair and vagina of the victim should be examined. The presence of spermatozoa in the vagina is proof of sexual intercourse, but not of rape; their absence is not proof that intercourse has not taken place, for they may have been removed by washing or by discharges or there may not have been an emission, or it may have been aspermic due to vasectomy or naturally. The presence of fresh seminal stains on the person or clothes of a woman provides strong evidence of intercourse, attempted or committed. Samples for seminal stains should be taken from the introitus and the perineum before visual and digital examination. The number of semen donors can be determined with the use of Y chromosome short tandem repeat markers.

(B) VENEREAL DISEASE: If the assailant is suffering from venereal disease, he may transmit it to his victim, which is strong corroborative evidence of intercourse. In gonorrhoea, an inflammation with an abundant mucopurulent discharge will be seen in two to four days (occasionally a week). In syphilis, an indurated ulcer on the external genitals may appear in about three weeks. Chlamydia infection, genital herpes, trichomoniasis, genital warts, hepatitis B and HIV may be transmitted.

(C) STRUGGLE: Signs of active resistance may be present. The fingernails may be broken or bent due to scratching the accused. Under the nails, debris may be present, e.g., blood, fibres, hair and skin fragment from the accused. Other signs of struggle may also be present.

(5) FINDINGS RELATED TO TIME OF ASSAULT:

(A) SEMINAL FLUID: The spermatozoa deposited in the vagina lose motility within 6 hours. Sperms may be recovered up to 24 hours from the vagina.

(B) VENEREAL DISEASE: The presence of venereal disease is useful in determining the time of sexual assault. The evidence collected by medical examination of the victim and accused, may either confirm or contradict the allegations made.

(C) WOUNDS: If the age of any wound present is consistent with the alleged time of rape, it will be of value.

SPECIMENS TO BE COLLECTED FROM VICTIM: The objects of collecting specimens are: (1) To obtain confirmation of the allegation. (2) To attempt to establish a link between the victim and the scene. (3) To attempt to establish a link between the victim and the assailant.

COLLECT: (1) Avulsed head hairs. (2) Pubic hair combings. (3) Avulsed pubic hair. (4) Matted pubic hair, if present. (5) Loose hairs found anywhere on the body. (6) Saliva for secretor grouping. (7) Swabs from bite marks for saliva. (8) Blood (plain) for grouping. (9) Blood (anti-coagulated) for alcohol and drugs. (10) Urine for drug screening. (11) Nail scrapings or nail cuttings for blood or tissue from other party. (12) Genital swabs from introitus, vagina and cervical os. Swab can be used to collect vaginal epithelial cells for DNA study. (13) Swabs from any soiled areas of skin. (14) Condom containing semen of the accused at the scene of crime or from the genitalia of the female.

Identity of the Accused: Blood, semen, urine, saliva, hair, and general debris present on the clothing or person of the victim may help the identity of the accused by comparing with the known materials from the accused. The presence of venereal disease is also helpful.

Summary: Evidence of rape is obtained from: (1) marks of violence on the body of the victim and the accused, (2) the presence of stains of semen, or of blood on the clothes and the body of the victim and accused, (3) marks of violence about the genitals, (4) the presence of seminal matter in the vagina, and (5) the existence of gonorrhoea or syphilis in both the parties. This evidence will vary according to circumstances. **Rape is not a medical diagnosis, it is only a legal definition. Medical proof of intercourse is not legal proof of rape. The report should contain negative as well as positive findings. The doctor should never make a diagnosis of rape. He may**

give opinion that there are signs of recent vaginal penetration, recent sexual intercourse, general physical injuries, and/or intoxication, and that the signs are consistent with the history given. Independent corroboration of the victim's story may be obtained by an eyewitness, evidence acquired at the crime scene, and from the body of the victim or accused. **Since many rapes are not witnessed, circumstantial evidence is offered in most cases.**

False Charges: Sometimes, false charges are made by a consenting woman, when the act is discovered by the parents or husband, when she becomes pregnant, or for purposes of revenge or blackmail. The parents may injure the genital organs of the child by introducing a blunt instrument or a thumb into vagina, or place irritants, such as chillies within the vagina to simulate rape. Adult women may stain their garments with a solution of starch or white of egg to simulate seminal stains and with the blood of an animal. In most cases, false charges are disproved by medical evidence, inconsistencies in the statement of the victim herself, to uncertainty as to consent, or to lack of corroboration. Undue delay in reporting the alleged rape is suspicious of the false allegations.

Complications: (1) Death may occur from (a) shock due to fright and emotion or by blunt force, (b) haemorrhage from injuries to genitals and perineum, (c) suffocation if mouth and nostrils are closed by the hand or cloth or by strangulation. (2) Mental derangements, convulsions and epileptic fits. (3) It may disrupt the victim's physical, social and sexual life.

Medicolegal Questions

(1) Rape and Resistance: In ordinary conditions, it is not possible for a man to have sexual intercourse with a healthy adult female in full possession of her senses against her will. The woman may not be able to offer marked resistance from terror or from an overpowering feeling of helplessness, or when her movements may have been obstructed by her clothing. The social status, physical development, and type of woman should also be considered. When a woman is overpowered by two or more men, she cannot resist much, and marks of violence may not be marked.

(2) Rape under Narcotics or during Sleep: Rape may be committed without the knowledge of the woman while she is under the influence of narcotics, such as opium, cocaine, hyoscine, alcohol, etc., while under the influence of an anaesthetic, while in a state of coma, and possibly while in a hypnotic trance. **The drugs associated with sexual assault are alcohol, rohypnol, (flunitrazepam) with other benzodiazepines, GHB, ketamine and scopolamine.** In a typical case, a sexual offender stealthily mixes the drink of an unsuspecting person with a sedative drug for the purpose of drugging and subsequently sexually assaulting the victim while under the influence of this substance. **Victims usually report loss of memory during and after these incidents. They wake up in unfamiliar places inappropriately dressed and often with the sense but not the actual recollection of having had sex.** The use of anaesthetic for surgical or dental operations may result in a charge of rape, especially in neurotic women, who in their anaesthetic flights of imagination believe themselves to have been sexually assaulted. There is no drug which can produce immediate unconsciousness when placed in front of the face of a person. It is impossible to have complete sexual intercourse with a woman during her natural sleep without her knowledge.

(3) Rape by Impersonation of the Husband: This is possible when the woman is sleeping. Rarely the victim may be awake, but lying in the dark.

(4) Rape by Fraud or Misrepresentation: Fraudulent sexual connections occur from time to time.

Rape Crisis Centres: They have been established in some countries. They provide (1) counselling to the victim and her relatives, (2) educate public on rape related issues and to reform the law, (3) reform the institutions which deal with the victims. These centres are largely staffed by volunteers, non-professional women, or who have been close to someone who was raped.

Child Molester: A child molester is a person who engages in any type of sexual activity with children. He can use violence or even murder to avoid identification. POCSO Act (Protection of Children from Sexual Offences Act) deals with these kinds of offences.

Female Circumcision: Sexual mutilation of small girls is done in some African countries involving excision of prepuce of clitoris, clitoris, labia minora or any combination of three. In India it is prevalent

among a most close-knit community done most secretively when the girls are 5 to 6 years old called Khatna. The community believes the clitoral head to be unwanted skin and a source of sin that will make women stay out of their marriage. The tradition aims to curb sexual drive in women and exert control.

EXAMINATION OF THE ACCUSED

General Procedure: If possible the accused should be examined by the same doctor who has examined the victim, for better correlation between the injuries found on the victim. The procedure of examination of the accused is similar to the victim. (1) Consent of the person should be taken in writing. It should be explained to him that the result of the examination may go against him and that he has the right to refuse consent. (2) The examination must be begun without delay as the signs of the act disappear rapidly. (3) He should be identified by the escorting police constable, and identification marks should be recorded. (4) A general medical history should include details of all past illnesses, surgical operations, serious accidents, medication and consumption of alcoholic drinks. (5) No attempt should be made to obtain any history of the specific incident. It should be found out whether the accused had consenting intercourse with any person within the previous 24 hours; when did he had the last bath; when was the last change of clothing and his explanations for the injuries found on the body. (6) The exact time, date, month and place of examination should be noted. (7) The age, development of genital organs and physical power of the accused should be noted and compared with that of female, to determine the possibility of his overpowering her. (8) His mental state and general behaviour should be observed. (9) If he appears to be under the influence of alcohol or drugs, it must be recorded.

General Examination: (1) The clothes should be examined for: (a) tears and loss of buttons, (b) hair, fibres and foreign matter, (c) cosmetic contact traces, e.g., face-powder or lipstick stains, (d) blood stains, (e) mud and other stains, grass, etc., and (f) seminal stains. (2) Nail scrapings should be examined for blood, epithelial cells, fibres, etc. (3) Note the matting of pubic hair due to emission of semen. (4) Female hair on the face, body or clothes of accused or of feminine pubic hair in his prepucial fold indicates commission of the offence. (5) Existence of venereal disease must be looked for. (6) Marks of struggle, such as bruises, scratches and teeth-bite marks on the face, neck, hands, thighs and genitals should be noted, caused during struggle from the resistance offered by the victim. The age of the injuries should be determined.

Genital Examination: (1) **Note the development of genital organs with special reference to the potency.** (2) Note any scratches or abrasions and bruising on genitals caused by the victim during struggle for prevention of intercourse. **Forceful penetration against resistance into a virgin hymen may produce tears or bruising of the frenulum of the prepuce in the uncircumcised and abrasion of the glans penis in both the uncircumcised and** the circumcised. Reddening of the glans especially of the rim, often patchy is more common. (3) Suspect's penis is washed with saline, and the material is stained with Papanicolaou's stain. **Vaginal and cervical cells and Barr body identification suggest recent intercourse**. (4) Examine the **glans for vaginal cells**. This is done by cleaning the organ with a filter paper and exposing the paper to vapours of Lugol's iodine. The paper becomes brown, if vaginal epithelial cells are present, because of the glycogen. The test is positive up to fourth day. (5) If accused is not circumcised, foreskin may be retracted and the presence or absence of **smegma on corona glandis** should be noted. **The absence of smegma (cheesy secretion of sebaceous glands, consisting mainly of desquamated epithelial cells) may indicate that intercourse might have been performed, but presence of smegma absolutely negatives possibility of complete penetration,** because smegma gets rubbed off during intercourse. Smegma will be removed by bathing or washing. **Smegma usually requires about 24 hours to collect**.

Specimens to be Collected from Accused: (1) Matted pubic hair. (2) Pubic hair combings and avulsed pubic hair. (3) Head hair. (4) Loose hair found anywhere on the body. (5) Nail scrapings. (6) Swabs from coronal sulcus, prepuce, penile shaft and urethral orifice, for they may reveal infecting organisms or blood that may also be present on the victim's swabs. (7) Blood for grouping.

INDECENT ASSAULT: Indecent assault is any **offence committed on a female, with the intention or knowledge to outrage her modesty.** Outrage means gross violation of decency, morality or feelings. Usually, the act involves the sexual parts of either or is sexually flavoured. In such assaults, a man may try

to kiss a woman, press her breasts, touch or expose the genitalia or thighs, try to put a finger in her vagina, play with vulva, etc. This is usually committed against children or old women. Stripping naked a female patient for medical examination is regarded as an assault.

Indecent offences between two or more male persons include such offences as friction of penis on the gluteal folds, handling of the male parts, mutual masturbation, etc., or intercrural connection (penile friction between the inner thighs and external genitalia). Men may encourage children to handle or masturbate their sexual organs. Such assaults are punishable under S. 354, I.P.C. up to one to five years imprisonment and fine. Abrasions or bruises may sometimes be present due to struggle.

SEXUAL HARASSMENT: It is an unwelcome sexual gesture or behaviour whether directly or indirectly as: (a) sexually coloured remarks, (b) physical contact and advances, (c) showing pornography, (d) a demand or request for sexual favours, (e) any other unwelcome physical, verbal, non-verbal conducts being sexual in nature.

INCEST: Incest is **sexual intercourse by a man with a woman, who is closely related to him by blood** (prohibited degrees of relationship), e.g., a daughter, granddaughter, sister, stepsister, aunt, or mother. Incest between father and daughter, and brothers and sisters are common. **These cases usually have psychological features. Incest occurs** (1) between mental defectives who are unable to understand the prohibitions against it, or whose feelings are too strong to inhibit them behaving in this way, (2) where alcohol removes the natural inhibitions, (3) in case of cerebral disease, such as general paralysis, senile cerebral degeneration, etc., (4) where a brother and sister have been separated since childhood or for a long period and meet later as strangers. It is not a punishable offence in India.

UNNATURAL OFFENCES

Voluntary sexual intercourse against the order of nature with any man, or woman, or animal is an unnatural sex offence (S. 377, I.P.C.). Penetration is sufficient to constitute the offence. These offences are punishable with imprisonment for life or up to 10 years and also with fine. **Homosexuality means persistent emotional and physical attraction to members of the same sex.**

SODOMY

Sodomy is the anal intercourse between two males or between a male and female. This used to be practiced in a town called Sodom. It is also called **buggery**. It is called **gerontophilia** when the passive agent is an adult, and **paederasty,** when the passive agent is a child, who is known as **catamite.** A **pedophile** is an adult who repeatedly engages in sexual activities with children below the age of puberty. Aggression and sadism are inherent components of paedophilia. The Greeks of the "Golden Age" also practised it, and it is sometimes referred to as **"Greek love"**. It can be heterosexual or homosexual. When practised between two men, they may alternately act as active and passive agents. **Any degree of penetration just into the anal margin, or any attempt at penetration is punishable. Proof of emission is not necessary.** When the act is performed with the consent of the passive agent, both are liable for punishment, but only the active agent is punished when the offence is committed without consent. It is difficult to be performed against the will, unless the person is drugged or very drunk, for slightest resistance is sufficient to prevent the offence. The crime is frequent among sailors, prisoners, in hostels, military barracks, etc., for they are thrown together for long periods. False charges may be made for blackmail, and men may be tricked into homosexual relationship by men disguising as women. This type of homosexuality is seen among all levels of the society.

Eunuchs: In India, a class of male prostitutes called **"eunuchs", act as passive agent in sodomy.** Among them there are two groups. (1) **Hijrahs**, and (2) **Zenana**, who live separately. **In hijrahs, penis and scrotum are removed before puberty by making a clean sweep with a knife at a secret ceremony.** The resulting scar becomes contracted and invaginated and is mistaken for female genitalia. They possess a typical feminine appearance, high pitched voice, abundance of fat round the hips, tendency to genu valgum, broad hips, absence of hair on body and face, and absence of genital organs. Their passions and expressions correspond to those of female, due to the resulting hormonal imbalance. **The genitalia of the zenana are intact.**

Passive Agent: General Procedure: Consent must be obtained before examination. A history of the circumstances and the nature of the act should be taken, especially as to the degree of penetration and use of the lubricant, etc. The examination must be carried out in the presence of a third person, either in lateral position or better in prone or in the knee-elbow position. Note the general appearance, feminine gestures, or clothing, and the presence of cosmetics, etc. A perianal and rectal swab should be taken first. The rectum should be examined with a speculum. General injuries together with scratches, especially on the back and buttocks should be looked for.

Anal Examination: Salient features: (1) Anal muscle is found dilated, irritable and tender to touch. (2) Digital examination may show loss of elasticity and tone. (3) Haematoma is frequently seen, either as a diffuse swelling of the anal margins, with obliteration of the normal anal skin folds, or as localised swelling. (4) **Linear abrasions, varying in number and extending from the anal margin into the anus itself, either anteriorly, posteriorly or laterally with adherent spots of blood suggest recent act.** (5) Anal fissures which may involve the external skin only, or may extend within the anal canal to the mucocutaneous junction. (6) If sudden violence is used, there may be triangular bruised tear at the posterior part of the anus. (7) Tearing of the sphincter ani is rare in adults and older children, but can occur in young children. (8) There may be anal prolapse. (9) Lesions are marked in children because of great disproportion in size between anal orifice of victim and penis of the accused. (10) Blood stains around the anus, on perineum and on clothes. (11) Faecal matter around the anus. (12) There is pain during walking, defecation and anal examination. For 2 to 3 days, there may be pain, congestion with oozing of blood stained fluid. (13) Lubricant matter, seminal fluid or venereal infections found at the anus or recovered by swabs from the rectum is a strong evidence of the crime. (14) Signs of struggle, e.g., bruises and abrasions, if he is not a consenting party (Fig. 17-2).

Diagnosis: The presence of a combination of semen, faeces, lubricant, and pubic hair on clothes is almost diagnostic of sodomy. The only proof of sodomy is the presence of semen in the anus.

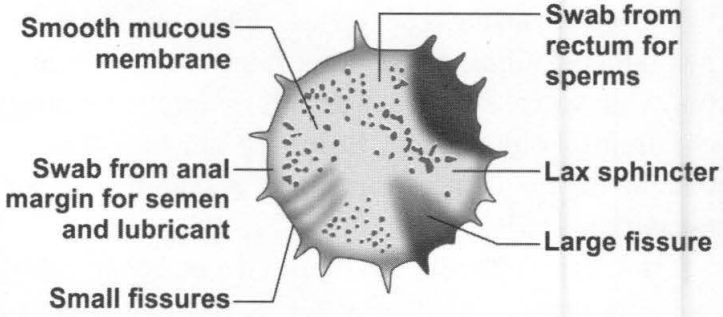


Fig. (17-2). Examination for evidence of sodomy.

Opinion: Opinion as to the cause of the dilation should be guarded and it should only be stated that it is consistent with entry of penis.

Habitual Passive Agent: The following signs may be present. (1) The shaving of the anal hair. (2) The skin about anus becomes smooth and thickened, extending up into the anal canal to the mucocutaneous junction. (3) Slight depression of buttocks towards the anus due to absorption of fat. (4) The muscle of the anus loses its tone and does not contract so readily when the skin around it is pinched. (5) Dilatation and laxity of the anus, and an epithelialization of the wrinkled mucosa of this part. (6) There may be anal fissures, scars, etc. (7) Absence of fine wrinkles in the anal mucosa. (8) Presence of venereal disease. (9) **Lateral buttock traction test:** A thumb is placed on each side of anus and lateral traction is applied. In a habitual sodomite, a complete relaxation of sphincter occurs with dilation of the opening which may be 4 to 5 cm in diameter through which rectum can be seen.

Specimens to be collected: (1) Head hair. (2) Pubic hair. (3) Loose hair and fibres found anywhere on the body. (4) Nail scrapings. (5) Swabs from any soiled areas of skin. (6) Swabs from the anal, perianal and lower rectum. (7) Blood. (8) Urine.

Active Agent: The routine medical examination of the accused should follow similar pattern as in the case of alleged accused in rape. (1) **The only evidence commonly found is the peculiar smell of anal glands transferred to the penis, and traces of faecal matter and lubricant on the organ.** (2) Abrasions on the prepuce, glans penis, tearing of fraenum or swelling or redness of penis. (3) Faecal soiling, blood and foreign hairs are likely to be found in the area of coronal sulcus. (4) In habitual sodomists, penis may

be elongated and constricted at some distance from the glans with twisted urethra. (5) Blood and seminal stains. (6) Presence of venereal disease. (7) Marks of violence on the body. (8) The clothes may show seminal stains or a mixture of semen and faeces.

BUCCAL COITUS (coitus per os or sin of Gomorrah): According to the Bible, this sin was common in a town called Gomorrah. In this type of sexual offence, **the male organ is introduced into mouth, of another male or female, usually of a young child.** Rarely, faint teeth marks and abrasions may be seen on the penis. Death may result from aspiration of semen, or impaction of the penis in the hypopharynx. The diagnosis is made by finding semen in the respiratory tract or stomach. **Buccal swabs should be taken which are useful up to only nine hours.** It is a punishable offence.

Male Prostitutes: The male prostitutes will submit to homosexual acts either as oral or anal inserters or receivers and even perform intercourse in the armpits (playing the bagpipes). The submammary fissure and intercrural folds are sometimes used.

TRIBADISM OR LESBIANISM: Female homosexuality is known as tribadism. According to Greek mythology, women of Isle of Lesbos practised this perversion. **Sexual gratification of a woman is obtained by another woman** by simple lip kissing, deep kissing, generalised body contact, manual manipulation of breasts and genitalia, genital apposition, friction of external genital organs, sucking of breasts or external genitals, etc. **In some cases, enlarged clitoris is used as an organ of passion or some artificial penis or phallus may be used.** The external genitals may show scratch marks, abrasions or teeth marks. **Many lesbians are masculine in type,** possibly because of endocrine disturbances, and are indifferent towards individuals of the opposite sex. The practice is usually indulged in by women who are mental degenerates or those who **suffer from nymphomania (excessive sexual desire).** It may lead to interference with young girls. Lesbians who are jealous of one another, when rejected may commit homicide, suicide or both. **Tribadism is not an offence in India.**

BESTIALITY: Bestiality is the sexual intercourse by a human being with a lower animal. The animals involved include those that are kept on the farm or as pets in households. Because of their convenient size, animals like calves and sheep are often involved. A few of larger birds like chicken, ducks and geese are also involved. Other animals used are cows, mares, she-asses and bitches. **Vaginal intercourse is the most frequent, but intercourse may take place through the anus or any other orifice, e.g.,** nose. This is seen in persons suffering from mental abnormality. Sometimes, the act is committed due to the false belief that gonorrhoea is cured by intercourse with a she-ass. Dogs and cats are the common animals for females. Usually, the animal manipulates the genitalia with its mouth, and actual coitus is very rare. Both the accused and the alleged animal are to be examined.

Signs in the Accused: (1) Animal faeces, vaginal secretion or hair, may be present on the penis. (2) There may be tearing of the freanum. (3) Presence of animal hairs, especially of its external genitals on the person or the clothes. (4) Stains of dung or animal blood on the person or clothes. (5) Marks of injuries on the body due to kicks, teeth or claws of the animal.

Signs in the Animal: (1) **Presence of human spermatozoa in the vagina or anal canal of the animal is a positive sign.** (2) Abrasions and lacerations with effusion of blood on the external genitals. (3) Presence of gonorrhoeal discharges in the animal.

SEXUAL PARAPHILIAS

Sexual paraphilias (sexual deviations, sexual perversions) are persistently indulged sexual acts, using objects or parts of the body in which complete satisfaction is sought and obtained without sexual intercourse. The mental state of the accused should be determined in all such cases by a psychiatrist.

URANISM: It is a general term for sexual paraphilias which includes sexual gratification by fingering, fondling, licking and sucking of the genitalia of opposite sex.

SADISM: (active algolagnia) (algos=pain; lognia=lust or craving) The term is derived from the name of a French nobleman, the Marquis de Sade, infamous for his crimes and writings. Many of his stories were about sexuality, cruelty and torture. In sadism, **sexual gratification is obtained or increased from acts of physical cruelty or causing of pain**

upon one's partner. It is seen more commonly in men. To obtain sexual pleasure, the sadist may bite, beat, whip, produce cuts, cigarette burns, etc. or ill-treat or torture his sexual partner in many other cruel ways. Extremely sadistic attacks may be made in which the victim's nipples may be bitten off, articles, such as a bottle, candle, or sticks, are inserted into the vagina, cigarettes or lighters may be used to burn the skin, and blows which may rupture internal organs or cause fractures of bones. Many are sociopathic, some schizoid, and others inadequate personalities.

LUST MURDER: **In extreme cases of sadism, murder serves as a stimulus for the sexual act and becomes the equal of coitus,** the act being accompanied by erection, ejaculation and orgasm. The typical lust murder is characterised by: (1) Periodic outbreaks. (2) Cutting or stabbing of the breasts, genitalia, or the lower abdomen, usually with sucking, licking or mouthing of the wounds and biting of the skin. (3) Erection and ejaculation may sometimes be followed by sexual intercourse with the dying or injured victim. (4) His behaviour is usually normal until the next outburst. Many rape murders result from an aggressive sexual reaction to inner fear. In true lust murders, mental disease is quite frequent. Every murder committed during a sexual act is not a lust murder. The murder may be the result of anger, jealousy, revenge, etc.

Algolagnia includes both sadism and masochism.

NECROPHAGIA: (necros = corpse; phagia = to eat): This is the extreme degree of sadism in which **the person after mutilating the body sucks or licks the wounds, bites the skin, drinks the blood, and eats the flesh of his victim to derive sexual pleasure.**

NECROPHILIA: (philia = to defile or foul): In this condition, there is a **desire for sexual intercourse with dead bodies**. It is said to have a sadomasochistic foundation and that decomposition, foul smell and coldness act as stimulants. There is also no danger of rejection or resistance. **The offence is usually committed on a newly buried corpse or a body awaiting burial.** The corpse may be mutilated following intercourse. Murder for the purpose of necrophilia is very rare.

Necrophilia and necrophagia are punishable under S. 297, I.P.C. with imprisonment up to 1 year.

MASOCHISM (passive algolagnia): The term is derived from the name of Leopold von Sacher Masoch, an Austrian novelist. Being whipped by his wife used to be a stimulant for his literary work. **This condition is the opposite of sadism.** In masochism, **sexual gratification is obtained or increased by the suffering of pain.** Masochists get pleasure from being beaten, abused, tortured, humiliated, degraded or dominated by their sexual partner, and they tend to place themselves repeatedly in self-defeating situations. Such painful stimuli may entirely replace the ordinary sex stimuli. It is usually found in males but it may be found in females, who may willingly expose themselves to the risks of severe bodily injury or murder at the hands of brutal husbands or lovers.

Sadism and masochism are rarely found in a pure state. They are usually found as a combination, with one type dominant over the other. **The combining of these practices is called bondage**. They are found in all age groups and in all socioeconomic levels. The acts of cruelty or pain associated with sadomasochism may serve as a stimulant for, or as complete substitute for sexual intercourse.

FETICHISM: **A fetish is an abnormal stimulus or object of sexual desire. Fetichism means the use of such objects for sexual gratification. In this, the person experiences sexual excitement leading to orgasm, from part of the body of a woman or some article belonging to her, that normally has no sexual influence on the mind, e.g., underclothing, brassiere, petticoat, stocking, shoes, etc., which act as a substitute for the female love object.** The fetish may be only incidentally associated with human body, e.g., a flower. In some cases, a picture of the fetish object provides sufficient stimulus. Sometimes, the act of stealing the article provides adequate sexual satisfaction, though often the fetish article is stored to the satisfaction of the fetish, or touching it gives him sex pleasure, or he may masturbate into the object. It is almost exclusively seen in males. They seldom commit violence or murder.

TRANSVESTISM OR EONISM: The term is derived from the name of Chevalier d'Éon Beamont, a Frenchman, who preached this. A transvestite (trans = opposite, vesta = clothing) is a **person whose whole personality is dominated by the desire to be identified and thought of as a member of**

the opposite sex. His dress, manner, occupational interests and associations are all designed to increase his feeling of being a woman. Sexuality with him is relatively unimportant, except as it promotes his feeling of feminity. There are varying degrees of transvestism. **It is usually found in the males who obtain sexual pleasure by wearing female dress.** Only small percentage is of homosexuals. Rarely, it develops out of a fetishistic interest in clothing or some part of opposite sex. Many cases are associated with sadomasochism. There is no hormonal disturbance or genital abnormality.

TRANSSEXUALISTS: They are persons who have an obsessional desire to become members of the opposite sex and seek surgery for anatomical alteration.

SEXUAL ORALISM: It is the obtaining of sexual pleasure from the **application of the mouth to the sexual organs.** It is seen both in heterosexuals and homosexuals. **Anilingus** (rimming) **is the kissing, licking and sucking of the anus by a sexual partner**. **Cunnilingus** (mouth job) **is the kissing, licking and sucking of a female's genitalia by her partner.** During these procedures there may be repeated thrusting of the tongue over the mandibular incisors which may cause injury to the tongue or ulceration of the lingual frenulum. **Fellatio** (irrumination; blow job) is the **stimulation of the penis by the partner's mouth.** Non-consensual fellatio may result in bruising (petechial and confluent) on the soft palate and at the junction between the soft and hard palates, laceration of the frenula and bruising and abrasion of the lips.

MASTURBATION: Masturbation (onanism; ipsation) **is the deliberate self-stimulation, which effects sexual arousal.** Mild masturbatory exercises are common both to men and women and are of little importance. In males, techniques are largely manual, by moving the penis against a bed or other object. Urethral insertions and anal stimulation and anal insertions are rare. Hollow articles, like bottles, test tubes, etc., are sometimes used, or articles made of rubber and plastic which simulate the female genitalia are used.

In females, a finger is gently and rhythmically moved over clitoris or labia minora, or steady pressure is applied over these parts with several fingers or whole hand. The genitalia may be rubbed against a pillow, a bed or some other object. Sometimes, women may insert fingers, wooden rods, glass tubes, metallic bars, bananas, etc., or artificial masculine genital parts made of rubber or plastic into the vagina. **Masturbation is an offence only when practised openly, e.g., in telephone booths, public lavatories, etc.**

EXHIBITIONISM: An exhibitionist is one who, over a period of six months or more, **experiences recurrent, intense sexually arousing fantasies, sexual urges, or behaviours involving the exposure of one's genitals to an unsuspecting stranger.** It usually occurs before adulthood. It is done mostly by males, often to children or the persons of the opposite sex. The pervert adopts a childish method of attracting attention to himself, to experience sexual gratification at the time of exhibition without physical contact. In some cases, the act is impulsive and spontaneous, while in other cases it is premeditated. Occasionally, women may expose themselves in public. Majority of them are psychopathic or suffer from compulsion neuroses. It is an obscene act, punishable under S. 294, I.P.C. with **imprisonment up to three months or fine.**

VOYEURISM OR SCOPTOPHILIA: It is the **counterpart of exhibitionism**. Voyeur (the so-called peeping tom) is defined as one who, over a period of six months or more, **experiences recurrent, intense sexual arousing fantasies, sexual urges or behaviours involving the act of observing an unsuspected person who is naked, in the process of disrobing, or engaged in sexual activity.** Most often, the victim is a stranger. Masturbation at the scene or later to the memories of watching the unsuspecting stranger is normally the source of sexual pleasure. The onset is usually before the age of 15 and may be chronic in nature. This perversion occurs in case of severe sociopathic personality disorder. To many males, observation of a female who is undressing may be erotically more stimulating than observation of a female when she is fully nude. Usually, such persons do not commit a major sex crime, but sometimes they may assault the victim or commit a murder. It is rare in females.

TROILISM: It is sexual practice involving 3 persons, 2 of one sex and one of the opposite sex. It is an extreme degree of voyeurism. **A perverted husband gets sexual satisfaction by inducing his**

wife to sexual intercourse with another man and by watching the same.

MIXOSCOPIA: It is a form of voyeurism in which **sexual gratification is obtained by the sight of others engaged in sexual intercourse.**

Oedipus Complex: It is sexual desire of son towards his mother. **Electra Complex:** It is sexual desire of daughter towards her father. **Pharoan complex** is sexual desire of brother towards his sister. Such persons are psychopaths with history of some mental trauma in the early life.

FROTTEURISM: Frotteurism (frottage; toucherism) **is contact with another person, in order to obtain sexual gratification.** Sexual satisfaction is obtained by **rubbing his private parts against a female's buttocks, thighs, breasts, etc., in crowds,** etc. If they attempt intercourse, they have a premature ejaculation or they are impotent. It is an uncommon perversion and rarely occurs alone. It is punishable under S. 290, I.P.C. with **fine up to 200 rupees.**

UNDINISM: In this, the sexual pleasure is often obtained by witnessing the act of urination by some one of the same or opposite sex. In some cases, pleasure is obtained by being urinated upon by the loved one or in urinating on him or her, but this is rare.

PYROMANIA: In most cases of pyromania, there is an underlying psychosexual disorder. Some have a latent form of sadism, and others obtain sexual stimulation or satisfaction while seeing the flames and destruction of a building.

Zoophilia: Sexual satisfaction is obtained from fondling of animals.

Bobbit Syndrome: This is a type of perversion in which the female partner amputates the penis of her male partner with a sharp-cutting weapon.

Scatalogia is associated with obscene telephone calls.

Pygmolionism is falling in love with an object made by him.

Narcism (Narcissism) is self-love, which may or may not include genital excitation.

Urolagnia: Sexual excitement is associated with the sight, or thought of urine or urination.

Caprolagnia: Sexual excitement is associated with sight or smell of faeces or defecation.

SEMINAL FLUID

Legal Issues: Seminal stains have to be detected in cases of rape or attempted rape, sexual murder of the female, sodomy and bestiality. Fertility of the fluid has to be proved in civil cases, e.g., disputed paternity.

Characteristics: Semen is greyish-yellow, thick, jelly-like and sticky when fresh. The quantity of seminal fluid in a single emission is two to five ml and contains about 60 to150 million sperms per ml of which 90% are motile at the time of ejaculation. The fluid is alkaline with a pH of 7.4. The stains are usually found on the clothing, but may be found on the person of either the victim or the accused. They may also be found on bed clothes, on floor or on the grass where the offence was committed. Seminal stains have to be differentiated from those due to starch, pus, leucorrhoeal discharge, and egg-albumen.

Collection of Material: (1) Fluid from the vagina is collected with a pipette or throat swab. (2) Dried or drying seminal fluid on the perineum or thighs is collected with a wet, throat swab. (3) The pubic hair should be removed and placed in a small container. (4) A portion of cloth containing the stain is cut out, dried and preserved. (5) Stains on smooth, impervious surface should be gently scraped off with the point of a knife into a glass container.

(I) PHYSICAL EXAMINATION: Seminal stains when dry have a greyish-white or yellow-grey colour and show an irregular, map-like outline. The cloth is stiffened as if starched. A fresh stain on a non-absorbent material appears translucent. After a month, it becomes yellow to brown. When examined under filtered ultraviolet light, they show a fluorescence of a bluish-white colour, which is not specific as other albuminous materials also fluoresce.

(II) Chemical Examination:

(1) Florence Test: The stain is extracted by one percent hydrochloric acid, and a drop is placed on a glass slide and allowed to dry. A coverslip is placed over this and a drop of florence solution (potassium iodide, iodine and water) is allowed to run under the coverslip. If semen is present, **dark-brown crystals of choline iodide appear immediately. They are rhombic crystals resembling haemin but are larger, arranged in clusters, rosettes, crosses, etc.** Choline originates from the seminal vesicles. The test is not proof of seminal fluid, but only of the presence of some vegetable or animal substance. A negative reaction is proof that the stain is not seminal.

(2) Barberio's Test: A saturated aqueous or alcoholic solution of picric acid when added to spermatic fluid, produces **yellow needle-shaped rhombic crystals of spermine picrate.** The reaction

probably depends on the presence of prostatic secretion.

(3) The Acid Phosphatase Test: The prostatic secretion element of seminal fluid contains 500 to 1000 times greater acid phosphatase than any other body fluid. Undiluted semen has an acid phosphatase activity of 340 to 360 Bodansky units or 2500 to 3500 Angstrom units per ml. The concentration of acid phosphatase gradually falls with time in vaginal secretions. **Semen recovered from the vagina retains recognisable acid phosphatase concentrations up to 36 hours after its deposition with gradual disappearance in 72 hours.** Concentration in excess of hundred Bodansky units with or without motile sperms indicates that ejaculation occurred within two hours of examination. **Dried seminal stains which have not undergone putrefaction retain acid phosphatase activity for weeks or months,** although enzymatic activity decreases slowly with time. In humans, acid phosphatase content is greater than in animals. This test is conclusive in the absence of demonstrable sperms or in aspermia.

(4) Creatine Phosphokinase: Spermatozoa contain a high concentration of creatine phosphokinase, which is more than double than found in any other body fluid. **Levels over 400 units of creatine phosphokinase per millilitre are almost diagnostic of seminal stains.**

(5) Immunological Method: Prostate specific antigen (PSA, P30) is a glycoprotein produced by prostate and is found in seminal plasma (normal and aspermic semen), male urine and blood, but not in any tissues or fluid of the female. **It is found in vaginal fluid up to 27 hours after sexual intercourse.** It can be detected in dried and old stains of more than 10 years in material stored at room temperature and in cadavers. Tests for P_{30} have replaced the quantitative analysis for acid phosphatase.

Fluorescent *in situ* hybridisation (FISH) has been suggested as a sensitive and specific test for detection of male epithelial cells in the post-coital vagina up to one week. Y-chromosome positive epithelial cells have been identified by vaginal swabs even in cases with no ejaculation.

(III) Microscopic Examination: A small piece of the stained fabric is moistened with a few drops of one percent hydrochloric acid, or three percent acetic acid in a watchglass for half to one hour when

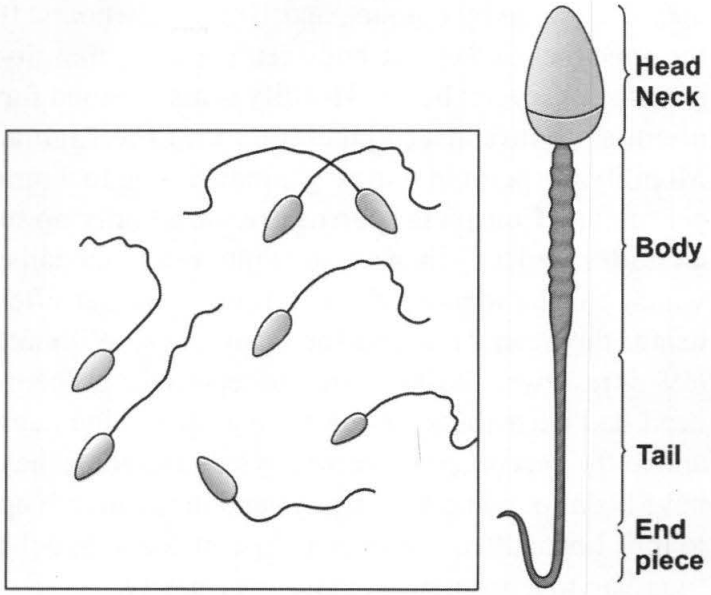


Fig. (17-3). Human spermatozoa

the stains are fresh, or two to four hours when old. Slides are prepared by rubbing the piece of fabric on them. Films are dried in the air without heat, or fixed for one minute in methyl or ethyl alcohol and then stained. The slide is stained with methylene blue for 15 to 30 minutes, and counter-stained with eosin for two minutes. The posterior half to one-third of head is stained deep-red or pink, while the anterior half to two-thirds appears unstained or is faintly stained with basic dye. The tail is stained pink. Old stains (even several years) may give positive results, but the older they are the less is the chance of finding intact sperms. **Human spermatozoa vary from 50 to 55 microns in length and consist of head, neck and tail.** The head is oval and flattened when seen in front and pear-shaped in profile. It is five microns in length and three and half microns in its greater diameter (Fig. 17-3). The neck is very short. The tail is long and tapered to a fine point. Spermatozoa are easily found in stains that dry rapidly. The head resists decomposition for some time, and as such, few heads of spermatozoa may be found in decomposed stain. They undergo disintegration within a few months, but may be found up to five years. With an ultrasound apparatus, maximum recovery of complete spermatozoa is readily obtained.

Motility of Sperms: A drop of mucus removed from vagina is placed on a glass slide, and diluted with a drop of normal saline, and examined for motile spermatozoa. When semen is kept at room temperature, full motility persists for about 3 hours;

50% are motile by 8 hours, and 10% for 24 hours. If the specimen is kept at body temperature, motility persists for several hours. **Motility is maintained for about six hours after ejaculation into the vagina.** Mobility of sperm in female genitalia is one to 4 mm per minute. **Complete sperms are found only up to 24 hours,** and they then separate into heads and tails, which can be identified up to four days, but after death, they can be found for many days. Willmott (1975) reviewed the literature and reported that sperm head had been found in the vagina up to nine days and in the cervix up to twelve days. In the anus, they have been found up to two days and in the mouth up to nine hours after intercourse. Spermatozoa may be found up to a fortnight in the uterine cavity.

Precipitin Test: The principle is the same as that for blood.

Group of Seminal Fluid: The specific agglutinable substances A and B, PGM, AK, HLA, LHD, Gm, Km, and GLO are present in the semen of secretors. As such, the group of the individual may be determined.

Proof of Semen: **The only absolute proof of semen is the finding of at least one unbroken spermatozoon, or electrophoretic LHD isoenzyme detection of sperms.** In the absence of spermatozoa, a stain which gives characteristic fluorescence in ultraviolet light, a high level of acid phosphatase, a high creatine phosphokinase and P^{30} can be considered to be due to semen.

Single photon fluorimetry has been used to differentiate between different semen. DNA amplification technique such as PCR and Y chromosome short tandem repeat (STR) markers can separate and identify different semens.

CHAPTER

18 Infant Deaths

Infanticide Act of England: According to the Infanticide Act of England (1938), **infanticide means the unlawful killing of a child under the age of one year. Only the mother can be charged with the offence, when the circumstances justify it, such as when the infant is killed by its mother while suffering from disease of the mind (psychotic disturbance or depression), due to the effect of stress, associated with her pregnancy, delivery, puerperium or lactation.** In such cases, the mother may not be held wholly responsible for killing the infant.

Law in India: In India, there is no such special Act, and as such **there is no distinction between the murder of newborn infant and that of any other individual.** **Foeticide** is the killing of the foetus at any time prior to birth. **Neonaticide** is the deliberate killing of a child within 4 weeks of its birth. **Filicide** is the killing of a child by its parents. **Infanticide does not include the death of foetus during labour, when it is destroyed by craniotomy or decapitation.**

Motive: Infanticide is rare, and usually committed by a young unmarried woman or widow. Infanticide is usually committed at the time of, or within a few minutes or hours after the birth. The alleged mother should be examined for signs of recent delivery.

STILLBIRTH: A stillborn child is one which is born after 28 weeks of pregnancy, and which did not breathe or show any other signs of life, at any time after being completely born. The child was alive in uterus, but dies during the process of birth. Stillbirths occur more frequently among illegitimate, immature male children in primiparae. The incidence is about 5%. It is common in obstructed labour due to contracted pelvis. It is born in sterile condition, and as such, putrefaction starts on the surface and extends inwards, whereas in case of newly born child which lived for some time, the bacteria inside the body may cause putrefaction to start in the abdomen. Lungs are dark-red. Squames and/or meconium within the alveoli does not indicate stillbirth. Meconium staining of the body merely indicates that foetal distress has occurred before or during delivery. **Signs of prolonged labour, i.e., oedema and bleeding into the scalp, and severe moulding of the head indicate stillbirth or death from natural causes shortly after birth.**

DEADBIRTH: A deadborn child is one which had died in utero and shows one of the following signs after it is completely born.

(1) **Rigor mortis at delivery**

(2) **Maceration: Maceration is a process of aseptic autolysis,** and is the usual change. **This occurs when dead child remains in the uterus for about three to four days surrounded with liquor amnii** but the exclusion of air. If air enters the liquor amnii after death of the foetus, putrefaction occurs instead of maceration.

Features: The earliest sign of maceration is reddening of skin with peeling, which can be seen in 12 hours after the death of the child in utero. Gas in chambers of heart and in aorta (in 12 hours) of foetus indicates foetal death (Robert's sign). The body of a macerated foetus is soft, flaccid and flattens out when placed on a level surface. It has a sweetish, disagreeable odour. The surface is slimy, blistered, desquamated and rarely jelly-like. Large blebs appear at 24 hours, which contain a red serous or serosanguineous fluid. The epidermis detaches easily and leaves moist and greasy areas (Plate 20-116). The tissues are oedematous. The abdomen is distended. The serous cavities may contain a turbid reddish fluid after 48 hours. The bones are flexible and readily detached from the soft parts. The joints become abnormally mobile. **All the viscera become soft and oedematous and lose their morphology, but lungs and uterus remain unchanged for a long time.** The umbilical cord is red, smooth, thickened and soft. **The skull bones are separated** and the brain has a greyish-red pulpy appearance. **Collapse of the vertebral column occurs.**

Spalding's sign: Loss of alignment and overriding of the bones of the cranial vault occurs due to shrinkage of the cerebrum after death of the foetus. In the early stage, there is only loss of alignment without overriding. The sign will develop earlier with a vertex presentation than with a breech. **It may be detected within a few days of death of the foetus,** but often takes much longer time, sometimes two to three weeks.

(3) Mummification: Mummification occurs when foetus dies from deficient supply of blood, when liquor amnii is scanty, and when no air enters uterus.

VIABILITY OF THE INFANT: Viability means the **physical ability of a foetus to lead a separate existence after birth apart from its mother, by virtue of a certain degree of development.** A child is **viable after 210 days of intrauterine life**, and in some cases after 180 days.

Livebirth: It means that the child showed signs of life when only part of the child was out of mother, though the child may not have breathed or completely born. The causing of death of such a child is regarded as murder.

SIGNS OF LIVEBIRTH

Legal aspects: In civil cases, any sign of life after complete birth of the child is accepted as proof of livebirth, e.g., hearing a cry, seeing movement of the body or limbs, muscle contraction, etc. A child may cry either in the uterus or in the vagina which may be heard by bystanders or even outside the room of delivery. This occurs only when the membranes have ruptured and air has entered the uterus. The law presumes that every newborn child found dead was born dead until the contrary is proved. In criminal cases, signs of livebirth have to be demonstrated by postmortem examination of the child.

Autopsy: (I) Shape of the Chest: Before respiration, the chest is flat and its circumference is one to two centimeter. less than the abdomen at the level of the umbilicus. **After respiration, the chest expands and becomes arched or drum-shaped.**

(II) The Position of the Diaphragm: The abdomen should be opened before the thorax, and the highest point of the diaphragm is noted, which is found about the level of fourth or fifth rib if respiration has not taken place, and **at the level of the sixth or seventh rib after breathing.** The position is affected by gases of decomposition.

(III) Lungs: Breathing causes important and permanent changes in the lungs, the extent of which depends on the physical strength and period of respiration **(Table 18-1)**.

(1) Volume: Unrespired lungs appear smaller, being collapsed on to the hilum when the thorax is opened. Fully respired lungs fill the pleural cavities, and the medial edges overlap the mediastinum and part of the pericardium.

(2) Margins: Before respiration the margins are sharp, which become rounded even when breathing is feeble. Glistening bullae appear along the margins when there has been a struggle to breathe due to some mechanical obstruction.

(3) Consistency: Before respiration the lungs are dense, firm and non-crepitant like liver. After respiration they are soft, elastic, spongy and crepitant. The lungs are also crepitant in putrefaction and after artificial inflation.

Table (18-1). Differences in lungs before and after respiration

Trait	Before respiration	After respiration
(1) Weight:	1/70 of body weight.	1/35 of body weight.
(2) Volume:	Normal or small.	Larger, and cover the heart.
(3) Consistency:	Dense, firm, non-crepitant.	Soft, spongy, elastic, crepitant.
(4) Margins:	Sharp.	Rounded.
(5) Colour:	Uniformly reddish-brown or bluish-red.	Mottled or marbled appearance.
(6) Air vesicles:	Not inflated.	Inflated.
(7) Section:	Little frothless blood exudes on pressure.	Abundant frothy blood exudes on section.
(8) Floatation:	Whole and parts sink in water.	Expanded areas or whole floats in water.

(4) Colour and Expansion of the Air-sacs: Before respiration, they are uniformly reddish-brown, or bluish-red, according to the degree of anoxia. The surface of the lobules is marked with shallow furrows. On section, the interior of lung is uniform in colour and texture, and little frothless blood exudes on pressure. After respiration, the air cells become distended with air. As the air vesicles expand, they become raised slightly above the surface, and may be seen as polygonal or angular areas on the surface of the lung, giving it a fine mosaic appearance. As the blood becomes aerated in the expanded area, the colour becomes a light red or pink, and the whole lung has a mottled or marbled appearance with rose-coloured patches of expansion and aeration alternating with the collapsed dark bluish-red areas. On section, frothy blood exudes from the cut surfaces on slight pressure. **Blood-stained froth in bronchi and bronchioles indicates that respiration has taken place.**

(5) Blood in the Lung Beds: The amount of blood in the lungs after respiration is about twice that in circulation before respiration.

(6) Weight: (A) Static Test or Fodere's Test: The lungs are ligated across their hila and separated. **The average weight of both lungs before respiration varies from 30 to 40 g, and after respiration from 60 to 66 g.** The increase in weight is due to the increased flow of blood.

(B) Ploucquet's Test: The blood flow in the lung beds is so increased after breathing that their weight is almost doubled, from 1/70 of the body **weight before respiration to 1/35 after respiration.** The increase in weight is not constant and is not a reliable indication of breathing.

(7) The Hydrostatic Test: It is based on the fact that **on breathing, the volume of the lungs is increased** which more than compensates the weight of the additional blood, due to which their specific gravity is diminished. **The specific gravity of the lungs before respiration varies from 1040 to 1050, and after respiration about 940.** A ligature is tied on the bronchi, and lungs separated. Each lung individually is placed in water. If they float, each lung is cut into 12 to 20 pieces and placed in water. If these pieces float, they are each squeezed in between thumb and index finger under the surface of water, to see if any bubbles of air escape, and if they still persist to float; or they are taken out of water, wrapped in a piece of cloth and squeezed by putting a weight to remove the tidal air. The pieces are again placed in water and if they continue to float, due to the presence of residual air, it indicates that respiration has taken place. If the pieces sink after pressure, respiration has not taken place. If some pieces float while others sink, it shows feeble respiration. It can simulate a false sense of scientific validity.

Fallacies of hydrostatic test: The expanded lungs may sink from: (1) Disease, e.g. acute oedema, pneumonia, congenital syphilis, etc. **(2) Atelectasis** (non-expansion) of the lungs due to: (1) Air not entering the lungs due to feeble respiration, but aeration of lungs may occur through the mucosa of trachea and bronchi. (2) Complete absorption of air from the lungs by the blood, if circulation continued after stoppage of respiration. (3) More air being expelled from the lungs during expiration than what is inhaled during inspiration. (4) Obstruction by an alveolar duct membrane.

The unexpanded lungs may float from: (1) Putrefactive gases. (2) Artificial inflation: The foetal lungs may be artificially inflated by blowing air through a tube, catheter or cannula passed into the trachea or by the mouth-to-mouth method. In such cases, the lungs can be inflated only partially and the stomach contains air.

Hydrostatic test is not necessary when: (1) The foetus is a monster. (2) The foetus is macerated or mummified. (3) The foetus is born before 180 days of gestation. (4) The stomach contains milk. (5) The umbilical cord has separated and a scar formed.

Respiration Before and During Birth: A child may breathe (1) while it is in the uterus, after the rupture of the membranes (vagitus uterinus), (2) **while its head is in the vagina** (vagitus vaginalis), (3) **while its head is protruding from the outlet.** A child which has breathed in the uterus or vagina may die from natural causes, before it is completely born. Therefore, **proof of breathing is not proof of livebirth.** In the newborn child, the respirations may not be strong or deep enough to expand the air cells and the child may live for some time on oxygen

absorbed from the respiratory cells of the alveolar ducts. Some air may pass into cells, but may not be sufficient to distend the fibrous tissue. This air may be absorbed by the blood, or may be lost. The child may live for many hours or even 1 or 2 days with only small portion of its lung tissue expanded. When the air cells have been distended once, they never return to the foetal condition. For the above reasons the hydrostatic test fails in a small percentage of cases.

Microscopic Examination of the Lungs: Microscopic examination is of value in determining the extent of respiration, and the presence of pulmonary disease or abnormality, which may have caused or contributed to the death. At full term, the normal foetal lung is almost completely atelectatic, but many of its terminal bronchioles and vesicles are partly expanded by amniotic fluid. This material is not stained with haematoxylin and eosin, giving the impression that alveoli have been well expanded by air. In a child who has breathed, diffuse atelectasis may be seen. It was thought that if respiration has not taken place, the alveoli appear as hollow gland-like structures lined by cuboidal or columnar epithelium, but if respiration has taken place, the alveoli are well-expanded and lined by flattened epithelium. This is not correct, for the changes in the type of cell which lines the air sacs does not occur with the onset of respiration. If the child has lived only for a few minutes, microscopy cannot always provide clear evidence of extrauterine respiration. Obstruction of the lower bronchial tree by hyaline duct membrane causes respiratory failure. The struggle to breathe may result in: (1) incomplete lung expansion, (2) suboxia and cyanosis, (3) petechial haemorrhages mainly subpleural, (4) oedema of the mediastinum and often of the lung.

(IV) Changes in the Stomach and Intestines: Air is swallowed into the stomach during respiration. The stomach and intestines are removed after tying double ligatures at each end. **They float in water if respiration has taken place, otherwise they sink. This is known as Breslau's second life test or stomach-bowel test.** This test is not of much value, because air may be swallowed by the child in attempting to free the air-passages of fluid obstructions in cases of stillbirth. It is useless when there is decomposition. **When dissected under water, the stomach shows mucus, saliva and air bubbles if respiration has taken place, and only mucus if breathing has not occurred.** Blood, meconium or liquor amnii in the stomach indicate that the child was alive at or shortly before birth. If milk is present in the stomach, it is positive evidence that the child has lived for some time after birth.

(V) Changes in the Middle Ear (Wredin's Test): Before birth, the middle ear contains gelatinous embryonic connective tissue. With respiration the sphincter at the pharyngeal end of eustachian tube relaxes and air replaces the gelatinous substance in few hours to five weeks. This is not at all reliable.

Livebirth is probable when: (1) All the lobes of the lungs are fully expanded. (2) There is oedema of the lungs, especially gross. (3) An alveolar duct membrane is present and has widespread distribution in the lungs. (4) Pulmonary atelectasis due to obstruction by an alveolar duct membrane is present. (5) Contusions of the lungs are present.

Stillbirth is probable in the presence of: (1) Maceration of the infant. (2) Flooding of the lungs with liquor amnii. (3) Desquamation of bronchial epithelium. (4) Distention of large bowel with meconium, indicating a struggle to breathe.

Changes after birth: Several changes occur in the child after birth, which are helpful in estimating the length of time the child lived after birth.

(1) Blood: Nucleated red cells usually disappear from the blood within 24 hours. Foetal haemoglobin (synthesised mainly in liver) which is about 80 to 90% before birth rapidly decreases to 7 to 8% at third month.

(2) Meconium: It is the green, sticky substance consisting of thickened bile and mucus. The meconium is completely excreted from the large intestine in the first 24 to 48 hours after birth, but in breech presentation and also in severe anoxia, the meconium may be excreted completely before birth. Meconium stains are brownish-green and stiffen the cloth. The reaction is acid.

(3) Caput Succedaneum: This is an area of soft swelling that forms in the scalp over the presenting part of the head in vertex presentations. The elevated, rounded area of the caput succedaneum corresponds to the portion of the scalp surface that is exposed within the opening of the dilated cervix during labour. **The scalp in the area of the caput is swollen to three to four times its normal**

thickness due to oedema and congestion, due to local interference with venous return produced by the pressure of the rigid cervical ring (Fig. 18-1). Most commonly, the caput succedaneum gradually diminishes within a week, often disappearing within 2 to 4 days after birth.

Cephalhaematoma: This is a localised accumulation of blood deep to the scalp, between the periosteum and bone surface. The haematoma is limited to the periosteal sheath of a single bone, commonly the right parietal bone and never crosses a suture line (Fig. 18-2). Cephalhaematoma is rare, **occurring in less than one per cent of newborns, and varies in size from 1 to 5 cm.** The haematoma swelling often tends to increase during the first day or two after birth, as more and more blood accumulates. **It shows the usual colour changes common with the bruises and disappears in about 15 days.**

(4) Skin: At first the skin is bright-red, which becomes darker on second or third day, then brick-red, yellow and normal in about a week. **Vernix caseosa covers** the skin, mostly in the axilla, inguinal region and folds of the neck, buttocks and persists for one or two days. Sometimes, it may be absent at birth and it is removed by washing. **Vernix caseosa is white cheesy substance consisting of sebaceous secretion and epithelial cells. It protects foetal skin against maceration while in liquor amnii.** The skin of the abdomen sheds in flakes during the first 3 days after birth.

Fig. (18-1). Caput succedaneum.

Fig. (18-2). Cephalhaematoma over the right parietal bone.

(5) Umbilical Cord: The blood clots in the cut end two hours after birth, and the vessels begin to be closed in about 24 hours. **The cord attached to the child shrinks and dries in 12 to 24 hours** (but this appearance is also seen in the body of a stillborn infant) and an **inflammatory ring forms at its base in 36 to 48 hours.** It contracts and mummifies on second or third day. Mummification of the cord also occurs after death if exposed to air. **The cord falls off on the fifth or sixth day and leaves an ulcer, which heals and forms a scar in 10 to 12 days.**

(6) Circulation: Contraction of the umbilical arteries starts in about 10 hours and are completely closed by third day. The umbilical vein and ductus venosus are closed on the fourth day. The ductus arteriosus closes by tenth day and foramen ovale by second or third month.

CAUSES OF DEATH: (I) NATURAL CAUSES: (1) Immaturity. (2) Debility due to lack of general development. (3) Congenital diseases, e.g., syphilis and disease of the child's internal organs, such as lungs, heart, brain, etc. (4) Malformations. (5) Haemorrhage from the umbilical cord, genital organs, stomach, rectum, etc. (6) Post-maturity. (7) Pre-eclamptic toxaemia. (8) Disease of the placenta or its accidental separation from the uterine wall. (9) Placenta praevia or abnormal gestation. (10) Neonatal infection. (11) Intrapartum or ante-partum anoxia. (12) Cerebral birth trauma. (13) Erythroblastosis.

(II) UNNATURAL CAUSES: These may be: (1) Accidental, and (2) Criminal.

(1) ACCIDENTAL CAUSES: (A) DURING BIRTH: (1) Prolonged labour: Severe compression of the head against contracted or deformed pelvis may cause intracranial haemorrhage and death with or without fissured fracture of the parietal bones. Subdural haemorrhages are common and usually bilateral. Tentorial tears may be bilateral and haemorrhage occurs into the subdural space either above or below the tentorium. In such cases, the head of the child shows evidence of well-developed moulding and caput succedaneum. Fractures and dislocations of the limb bones and clavicles may be found.

(2) Prolapse of the cord or pressure on the cord produces death by asphyxia. The cord is liable to be compressed by the foetal head, especially in breech presentations.

(3) Twisting of the cord round the neck or knots of the cord cause strangulation.

(4) Injuries to the Mother: Heavy blows or kicks on the mother's abdomen or falls from height, may cause concussion of the brain of the child, with or without fracture of skull or rupture of blood vessels or organs. Rarely, powerful uterine contractions may fracture the cranial bones of the foetus.

(5) Death of the Mother: The child can be saved, if it can be delivered within 5 to 10 minutes of the mother's death.

(B) AFTER BIRTH: (1) Suffocation: It may result when the membranes cover the head during birth, or if the face is pressed accidentally in the cloth or submerged in the discharges, such as blood, liquor amnii or meconium. A child can survive in the membranes for 20 to 30 minutes.

PRECIPITATE LABOUR: Labour terminating in a very short time than that taken on the average, either by a primipara or multipara is called precipitate labour. In this, delivery occurs suddenly and rapidly without the knowledge of the mother. All the three stages of labour are merged into one. The foetus is normal or premature. **It is possible in multiparae with large roomy pelvis, but is extremely rare in primiparae.** Sometimes, a woman may not be able to distinguish the sense of fullness produced by the descent of a child, from the feeling of bulky evacuation. **The child may die from (1) suffocation** by falling into a lavatory pan, (2) **head injury** and fracture of the skull with subdural haemorrhage often bilateral, by a fall on a hard floor, if the woman was standing, and (3) **haemorrhage** from the torn end of the cord.

If the birth occurs into the toilet bowl, or into a bucket containing liquid, the infant will inhale the liquid and blood, and meconium and vaginal mucus are found in the air-passages. In accidental falls, the haemorrhages are usually subdural and often bilateral. The average length of the cord is 50 cm. which is not sufficient to allow the child to fall to the ground, and is sufficiently strong to withstand the weight of the foetus without breaking. **The cord is torn most commonly at the foetal end than the placental end, but is not torn in its middle, or the placenta is expelled with the child.** The [illegible] **absent.** Foreign materials, such as mud, sand, gravel, may be found in the hair or injured scalp of the child. **The fractures of the skull are usually fissured and limited to parietal bones,** but may extend to frontal and squamous part of temporal bones.

Medicolegal Importance: (1) The mother or her relatives may be accused of killing the infant, while the death may be due to injury, haemorrhage or asphyxia from precipitate labour. (2) In a case of infant murder, death may be attributed to precipitate labour.

(2) CRIMINAL CAUSES: (A) ACTS OF COMMISSION: They are acts done positively to cause the death of the infant.

(1) Suffocation, (2) Strangulation, (3) Drowning, (4) Burning, (5) Blunt Head Injury, (6) Fractures and dislocations of cervical vertebrae, (7) Wounds, (8) Poisoning.

(B) Acts of Omission or Neglect: A woman is guilty of criminal negligence, if she does not take ordinary precautions to save her child after birth. The following acts of omission amount to crime. (1) Failure to provide proper assistance during labour may cause death by suffocation or head injury. (2) Failure to tie the cord after it is cut may cause death by haemorrhage. (3) Failure to clear the air-passages which may be obstructed by amniotic fluid or mucus. (4) Failure to protect the child from exposure to heat or cold. (5) Failure to supply the child with proper food.

THE ABANDONING OF INFANTS: If the father or mother of child under the age of 12 years, or anyone having the care of such child, leaves such a child in any place with the intention of abandoning such child, shall be punished with imprisonment up to 7 years [S. 317, I.P.C.]. If the child is a girl, rigorous imprisonment may extend to 10 years and also fine extending to one lakh rupees.

CONCEALMENT OF BIRTH: Whoever, by secretly burying or otherwise disposing of the dead body of child, intentionally conceals the birth of such child, shall be punished with imprisonment up to two years (S. 318, I.P.C.). If the child is a girl, rigorous imprisonment may extend to five years and also fine extending to fifty thousand rupees.

BATTERED BABY SYNDROME OR NON-ACCIDENTAL INJURY OF CHILDHOOD: It is also known as child abuse syndrome, Caffey's [illegible]

A battered child is one who has received repetitive physical injuries as a result of non-accidental violence, produced by a parent or guardian. In addition to physical injury, there may be non-accidental deprivation of nutrition, care and affection. **The classical features of syndrome are obvious disagreement between the nature of the injuries and explanation offered by the parents, and delay between the injury and medical attention, which cannot be explained. The constant feature is repetition of injuries at different dates, often progressing from minor to more severe.**

Features: (1) Age: Usually less than 3 years. **(2) Sex:** Slightly more in males (55 to 63%). **(3) Position in family:** One child of a family, commonly the eldest or the youngest and often unwanted, such as the result of pregnancy before marriage, failure of contraception or an illegitimate child. **(4) Socio-economic factors:** Parents tend to be young between 20 and 30 years, and belong to lower social class and lower education. The family is usually isolated. There is often a history of family disharmony, long-standing emotional problems, or financial problems. Many mothers have multiple social and psychiatric problems. The mother is of lower intelligence quotient, (I.Q.), often pregnant or in the premenstrual period at the time of battering. Most of the parents suffer from guilt-amnesia. **(5) History:** There is obvious difference between the nature of the injuries and the explanation offered by the parents, which may change on several times of repetition, each time the child is taken to a different doctor. **(6) Treatment:** There is always unexplainable delay between the injury and medical attention.

INJURIES: Soft tissue injuries are very common. Laceration of the mucosa inside the upper lip, often tear of fraenulum, is the most characteristic lesion. Multiple bruises of various ages all over the body from rough handling, or beating, kicking or throwing the infant are common. Bite marks may be found on the cheeks, shoulders, chest, abdomen, arms, legs and buttocks. Bruises are usually present around the elbows and knees due to gripping of the child, so as to shake or pull him, or hurl him into cot or against furniture, etc. Subgaleal haematoma resulting from vigorous pulling on the scalp is characteristic.

Visceral injuries: Subdural haemorrhage is found in about 40% of fatal cases. Crushing or compressing forces applied to the abdomen produce either "bursting" injuries of the liver or spleen or perforations of distended hollow viscera including the stomach, intestine or urinary bladder. Small circular, pitted burns may indicate deliberate stubbing of cigarette ends upon the skin. The violent forces applied to the limbs involve pulling and twisting, both capable of producing epiphyseal separation and periosteal shearing. Long bone fractures result from compression, bending and direct forcible blows. Anteroposterior compression of the chest causes fractures of ribs in midaxillary line. Violent squeezing of the chest from side to side causes fractures at the costochondral junctions. Multiple rib fractures also occur along the posterior angles of the ribs. After about 2 weeks, callus is formed, and on X-ray a "string of beads" appearance is seen in the paravertebral gutter (nobbing fractures). Before autopsy, a whole body X-ray should be taken to detect old fractures.

Diagnosis: The diagnosis depends upon (1) Nature of injuries. (2) Time taken to seek medical advice, and (3) recurrent injuries. Differential diagnosis has to be made from scurvy, congenital syphilis, infantile cortical hyperostoses and osteogenesis imperfecta.

MUNCHAUSEN'S SYNDROME: Munchausen's syndrome refers to adults who injure themselves or feign signs of illness, and go from hospital to hospital for unnecessary investigations and treatment. These patients appear to be compulsively driven to make their complaints. **The person is aware that he is acting an illness, but he cannot stop the act.** There is continuity, ranging from exaggerated claims of infirmity to actual self-induced illness.

MUNCHAUSEN'S SYNDROME BY PROXY: It is a variant of Munchausen's syndrome, which is a very peculiar and dangerous type of child abuse usually involving the mother in more than 90% cases, in which children are brought to doctors for induced signs and symptoms of illness with a fictitious history. It has been described in children of few weeks of age to 21 years, but is most common in the first two years of life. **More than 50% mothers have personal abnormal illness behaviour in the form of fictitious or somatoform disorder.** Often the parental illness alternates with that of the child; the self-injurious behaviour of the parent may wax and wane as the fictitious illness of the child waxes and wanes. The child is admitted frequently in the hospital for medical evaluation for the non-existent conditions.

Method of simulation or production of illnesses: (1) The mother pricks her finger and adds blood to the urine of the child and takes the sample to the doctor. (2) The mother gives insulin to the child and takes to hospital with hypoglycaemia. (3) The child's nose is closed with two fingers and the lower jaw pushed up with the palm to block the airway. (4) A pillow or towel is put over the face of the child and the face is pushed down into bed clothing. (5) Vomiting: allegation or by ipecacuanha. (6) Diarrhoea: laxatives or salt poisoning. (7) CNS depression: barbiturates, benzodiazepines. (8) Fever: alleged. (9) Rash: scratching or intoxication.

Fabrication may impact child's physical and emotional development.

SUDDEN INFANT DEATH SYNDROME: Sudden infant death syndrome **(SIDS), or cot death or crib death is defined as the sudden and unexpected death of a healthy infant, whose death remains unexplained even after a complete autopsy.**

Features: (1) **Incidence:** 0.6/1,000 livebirths. (2) **Age:** Two weeks to two years. Majority of cases occur between one month and 7 months with a marked peak between two to three months. (3) **Sex:** There is slight increase in the males. (4) **Twins:** There is increased risk (three-fold) amongst members of a twin pair. Most twins are premature and of low birth weight. (5) **Geographical distribution:** The occurrence is worldwide. (6) **Time of Death:** Death always occurs during sleep at all times of night with a moderate increase in the early morning hours. (7) **Socioeconomic standard** of the family is usually low.

The child is either quite well when put to the bed, or may have only a minor upper respiratory tract infection (cold or snuffles), or minor gastrointestinal disturbances. Cot deaths are major causes of death in infants in the first 6 months of life.

Autopsy: Milk or a bloodstained froth is sometimes seen on the child's mouth, nostrils, or bedding. The postmortem findings are negative. In about 15% of cases, some pathological condition may be found, such as frank pneumonia, congenital heart disease, Down's syndrome or a tracheobronchitis. The only constant findings are multiple petechial haemorrhages on the visceral surfaces of the heart, lungs and thymus which are agonal in nature, perhaps from terminal respiratory efforts against closed glottis. Bloodstained fluid at the mouth and nose is present in about 50% cases. Peribronchial cell infiltration is the main finding. A small amount of milky vomit in the trachea and main bronchi and shedding of individual tracheobronchial epithelial cells are commonly found. Many infants show froth in the air-passages and facial pallor. The lungs show patchy or uniform purplish discolouration of the surface and are firm in consistency with congestion, oedema and increase in weight. **It is a diagnosis of exclusion.**

Theories: There is no single cause of cot death, and death may result from a number of causes which combine and act via a common pathway of cardiorespiratory failure, while the child is passing through a vulnerable period of development. Probable mechanism of deaths may be due to electrolyte imbalance, conduction disorders, respiratory distress, metabolic disorders, overlaying, viral infection, prominent tracheobronchial secretions. Whatever the cause, factors in pregnancy that inhibit foetal circulation could damage the child's brain, so that it no longer controls breathing properly. An unidentified trigger could affect the airway of the sleeping infant. The brain would not respond correctly and the breathing would stop.

CHAPTER

19

Blood Stains

Blood-stained article is allowed to dry at room temperature. Extra heat should not be used as this will cause deterioration of the stain. If the stained clothes are not dried, putrefaction sets in and it becomes difficult or impossible to know whether the blood is of human or animal origin. **All stains should be sent to the State Forensic Science Laboratory.**

Collection of Blood Stains: (1) A clean piece of white filter paper, gauge or cloth may be used, allowing blood to soak into it, and then drying it at room temperature. (2) If the object is porous, a portion of unstained area should also be taken if this is practical. (3) If the object is non-porous and particularly if it is metallic, stains can be removed by scraping and placed in small glass containers. (4) Stains on clothing may be scraped off or a fragment of the material cut. (5) Liquid blood can be pipetted and placed in a bottle and refrigerated.

Solvents: The solvents for blood stains are: (1) 10% solution of potassium cyanide, (2) 10% solution of glycerine in distilled water, and (3) a weak solution of ammonia.

SUBSTANCES RESEMBLING BLOOD STAINS: Rust stains, synthetic dye stains, mineral stains, some vegetable stains and spots of grease, resin, tar and pitch, especially on dark fabrics may resemble old blood stains.

STAINS ON CLOTHING: In the case of clothing, type of garment, its colour and consistence should be noted and if the garment is torn, the position of the tears should be noted. Both the outer and inner surfaces of the garments should be examined. The position of all stains should be given correctly by a description of the stain in its relation to the manner in which a garment is usually worn, e.g., a stain on the trousers should be described as being above, behind, or to the outer side of the knee. Stains may also be described in relation to the pockets, the buttons or the seams of a garment. The size and the shape of the stain should be noted. If the stain is in the form of a smear, its general direction should be noted. Blood stains are extremely resistant to washing by water. Dried blood on a dead body or article is very resistant for quite a long time even though the body has been totally submerged. Invisible blood stains can be detected by spraying luminal on the cloth or stained material with an atomiser inside a dark room. The stained area will luminesce if blood is present in those areas.

EXAMINATION OF BLOOD STAINS

(I) GENERAL EXAMINATION: (1) SCENE OF THE CRIME: When much blood is present it suggests serious injury during life, but if a large vessel is cut, bleeding can occur after death. The collection of a pool of blood near the body during life indicates that the deceased fell unconscious and remained immobile after the injury. A trail of blood stains will indicate that the victim was wounded at some distance from the place at which the body is found. It can happen when the victim is attacked while running or in case of suicide. Blood coming from the arteries of a living person will be scattered in fine spray over the surface upon which it has fallen. Venous bleeding is a slow steady flow, causing a pool if the victim is at rest, and separate widely spaced drops, if the victim walks about.

The direction of the fall of blood on to a surface may be recognised. If it drops vertically on to flat surface, the stains are circular. If the height does not exceed a few centimetres, the drop appears as a round spot. If it has travelled 30 cm. or more, it shows prickly edges, the projections growing finer and larger in number with the increase in length. When the height is still greater ray-like splashes break out from the drop, and may be seen up to a distance of 20 cm. Splashes of blood striking a surface obliquely may appear like spears or exclamation marks; the pointed end indicates the direction of the motion. When blood falls upon porous articles or clothing, such as linen or cotton, it is absorbed and spreads. Smears caused by fingers or palms are helpful in identification (Fig. 19-1).

(2) PART OF THE BODY FROM WHICH STAIN IS DERIVED: Menstrual blood is usually found on female garments, diapers or pieces of cloth. It is dark and fluid, has a disagreeable smell and the reaction is acid.

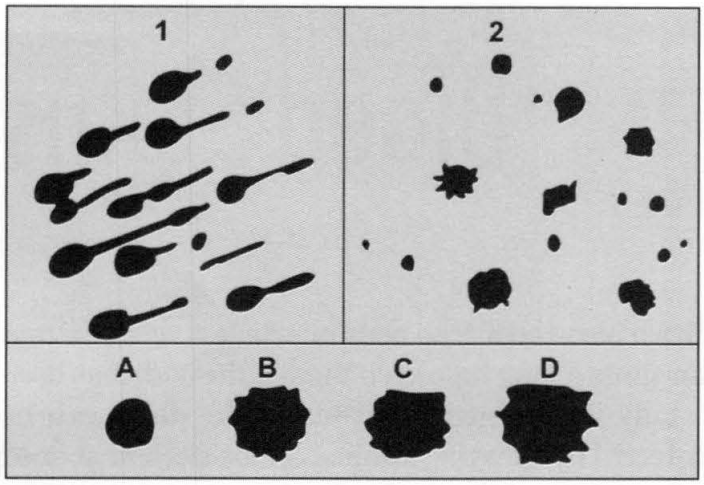


Fig. (19-1). (1) Spurting (indicating direction). (2) Blood falling vertically. (3) Drops of blood from various heights. (A) Up to 1.5 metres. (B) 1.5 to 3 metres. (C) 3 to 5 metres. (D) 5 to 7 metres.

On microscopic examination, it shows endometrial and vaginal epithelial cells and number of microorganisms. It contains fibrinolysins. If the blood is from the nose, mucus and hair from the nose may be found. Vomited blood is of chocolate colour and acid in reaction due to the action of gastric juice. Blood due to haemoptysis is bright-red and frothy, with alkaline reaction. In blood due to rape, semen and pubic hair may be found. Blood stains due to boils and sores show a smeared appearance without definite drops of blood, and may contain pus cells and bacteria.

(3) AGE OF BLOOD STAINS: Fresh stains on light-coloured clothes are of bright-red colour, which gradually changes to reddish-brown in 24 hours and brown within a few days, which may become black after a long time. Fresh stains are moist and sticky, and on drying, they stiffen the cloth because of the proteins. On many metallic articles, blood stains appear as dark shining spots or smears and when dry show fissures and cracks. In ordinary conditions, a drop of blood dries in an hour or two. The recently shed arterial blood is bright-red and venous blood dark-red. The fresher the blood, the more easily it is dissolved in water. Fluorescence decreases as the stain becomes older. It can only be stated that the stain is very fresh, recent, some weeks or months, or very old.

(4) SEX AND AGE OF PERSON: Sex can be determined from the presence of sex chromatin (Davidson body) in the leucocytes, if the cells can be identified. At birth, the blood forms a thinner and softer coagulum. The presence of foetal haemoglobin indicates that the blood is derived from an infant.

(5) LIVING OR DEAD BODY: Blood which has effused during life can be removed in scales on drying, due to the presence of fibrin. Blood which has flowed after death tends to break up into a powder on drying.

(6) SOURCE OF BLOOD: If the victim and assailant are of different blood groups, it is helpful in establishing the identity. If the stains are on the inner side of the garment, they usually belong to the victim, but if found on outer side they may belong to the victim or accused.

(II) CHEMICAL EXAMINATION: The chemical tests depend on the presence in the blood stains of an enzyme peroxidase, which in the presence of hydrogen peroxide, oxidises the active ingredient of reagent and produces the characteristic coloured compound.

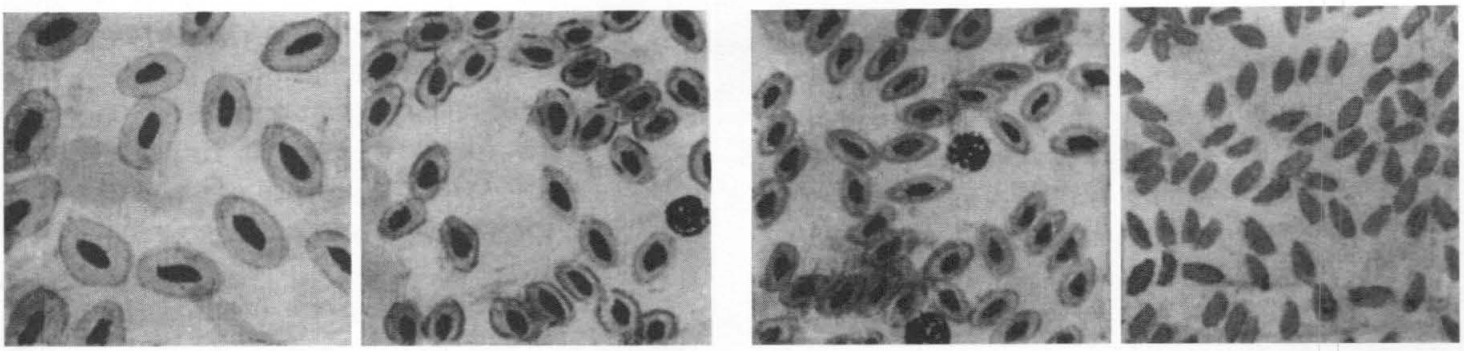
(1) BENZIDINE TEST: Cut out a small piece of stained material or tease out fibres from the stained fabric and place it on porcelain tile. Add a drop of saturated solution of benzidine in glacial acetic acid, and then a drop of 10 volumes hydrogen peroxide. If blood is present, dark-blue colour is produced immediately.

A positive reaction is given by blood of almost any age, blood that has been exposed to heat or cold and blood stains treated with cleaning agents. This is the best preliminary test for blood and it detects blood when present in a dilution of one part of blood in three lakhs. A positive reaction is not proof of the presence of blood, but a negative reaction rules out the presence of blood. A weaker reaction is obtained from certain other substances, e.g., pus, saliva, milk, rust, formalin, certain vegetable and animal juices, oxidising agents, bacteria, etc.

(2) PHENOLPHTHALEIN TEST (KASTLE-MEYER TEST): To a solution extracted from the stain with distilled water, add 10 to 20 drops of phenolphthalein reagent and then a drop or two of 10 volumes hydrogen peroxide. If blood is present a pink or purple colour develops immediately. The test is extremely delicate and detects blood in a dilution of one part in five millions. Traces of copper give positive reaction.

The tests employing guaiacum (deep blue) and leucomalachite green are rarely used in medicolegal work.

(III) MICROSCOPIC EXAMINATION:
(1) Red Corpuscles: Intact red cells are seen only when the stains are fresh, or when a fragment of clot is available. The red cells become unrecognisable when dried. A small piece of stain is cut out and soaked in a watch glass with two to three drops of Vibert's fluid (sodium chloride 2 g., mercuric



Frog Fish Hen Camel

Fig. (19-2). Red blood corpuscles.

chloride half g., distilled water 100 ml.), or normal saline for half-an-hour. It is then teased with needles and examined under high power. **Red blood cells are circular, biconcave, non-nucleated discs in all mammals except camels. In camels they are oval and biconvex but non-nucleated. In birds, fishes, amphibia and reptiles they are oval, biconvex and nucleated** (Fig. 19-2).

(IV) MICROCHEMICAL TESTS: **They are based on the property of haeme (iron) part of haemoglobin to form characteristic coloured crystals with certain reagents.**

(1) Haemin Crystal Test (Teichmann's Test): A small crystal of sodium chloride and 2 to 3 drops of glacial acetic acid are placed on a small piece of the suspected stain on a glass slide. A coverslip is applied and the acid is evaporated by heating over a small flame. It is examined under the microscope after cooling. **Faint yellowish-red to brownish-black rhombic crystals of haemin or haematin chloride, arranged single or in clusters are seen if blood is present** (Plate 20-117).

(2) Haemochromogen Crystal Test (Takayama Test): Place a small piece of suspected material on a glass slide and add 2 to 3 drops of Takayama reagent (sodium hydroxide, pyridine, glucose) and cover with a coverslip. **Pink, feathery crystals of haemochromogen or reduced alkaline haematin arranged in clusters, sheaves, etc., appear in 1 to 6 minutes** (Plate 20-118). Slight warming of the slide hastens the reaction. The test gives good result even with old stains. It is delicate and more reliable.

(V) SPECTROSCOPIC EXAMINATION: It is the most delicate and reliable test for detecting the presence of blood in both recent and old stains. Less than 0.1 mg. of blood is sufficient. The bloodstain is dissolved in water, normal saline or dilute ammonia, and is placed in a small glass test tube, which is then put between the spectroscope and the source of the light. The extract of the blood must be dilute and if turbid it should be filtered. The solution of the blood has the property of absorbing some of the rays from the spectrum, producing characteristic dark absorption bands, which vary with the type of the blood pigment present.

SPECTRA OF HAEMOGLOBIN AND ITS DERIVATIVES (FIG. 19-3): **(1) OXYHAEMOGLOBIN is marked by two distinct bands in the yellow between the Fraunhofer lines D and E, the one nearer D being about half the breadth of the other and more defined.**

(2) REDUCED HAEMOGLOBIN shows a broad band which lies between D and E.

(3) CARBOXYHAEMOGLOBIN has a spectrum similar to oxyhaemoglobin. Carboxyhaemoglobin remains unchanged after addition of ammonium sulphide, which reduces oxyhaemoglobin.

(4) METHAEMOGLOBIN: Spectrum is similar to oxyhaemoglobin with a third dark band in the red between C and D and the fourth between E and F, which is more indistinct.

(5) ACID HAEMATIN has a sharp band between C and D and a broad band between D and F which is not well-defined.

(6) ALKALINE HAEMATIN has a band between C and D.

(7) HAEMOCHROMOGEN or reduced alkaline haematin has a dense narrow band midway between D and E and a pale, broad band over E.

(8) CYANHAEMOCHROMOGEN has absorption bands, similar to those of haemochromogen, but slightly wider.

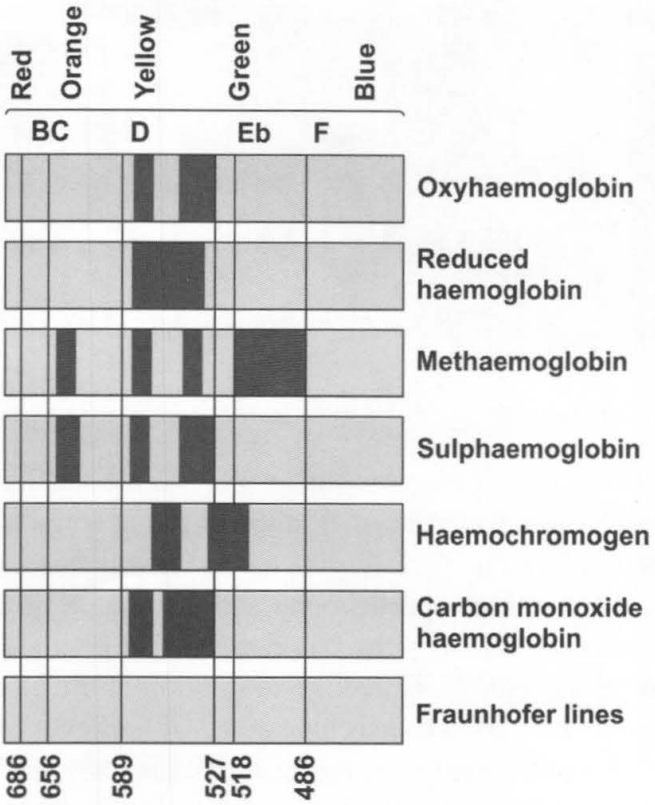


Fig. (19-3). Absorption spectrum of haemoglobin and its derivatives.

(9) ACID HAEMATOPORPHYRIN gives a dark sharp and broad band between D and E.

(10) ALKALINE HAEMATOPORPHYRIN consists of four bands. One between E and F is darkest and broadest, two are between D and E and the fourth between C and D.

Forensic Serology: It involves examination and analysis of a variety of blood fluids which includes blood, saliva, semen and urine.

(VI) SEROLOGICAL EXAMINATION: This determines whether the blood is derived from human being or from a lower animal.

(1) Precipitin Test: Principle: Blood serum contains protein in colloidal suspension, and when human serum is injected into an animal, the animal becomes immunised against these proteins, and antibodies develop in its blood. **If human serum is then brought into contact with this animal serum, the antibodies in the animal serum react with the proteins in the human serum, and a visible precipitate forms.** The antibodies causing this reaction are known as precipitins, and the animal serum is known as an anti-human precipitin serum. A rabbit or a fowl is injected with human blood every third day for three to five injections. After this the animal is killed, and the antiserum is collected. A suitable antiserum should react immediately or within a minute on the 1:1,000 dilution.

Technique: The presence of blood is determined first. An extract of stained material is prepared by soaking it in normal saline. No chemical should be added to extract the stain. The extract should be clear and may be filtered or centrifuged if necessary. The extract is diluted to 1:100 with normal saline. **Two drops of undiluted antiserum are added to 0.75 ml. of diluted stain extract in a small tapering test tube held in a slanting position. The antiserum slowly settles down to the bottom, and at the junction of two fluids a white ring with well-defined borders appears in the case of a positive reaction.** The ring is situated mostly in the antiserum. In the case of negative reaction no ring appears. A positive reaction should begin in 10 minutes and should be read in 30 minutes. **The results are qualitative and expressed as positive or negative.** For medicolegal purposes, all doubtful reactions are read as negative.

Application of the test: It is a specific protein test, and the reaction demonstrates the presence of albuminous substances obtained from any part of human body. The origin of skin, flesh, bone or even secretions, such as saliva, milk and semen is determined by this test. It is a crude test.

(2) GEL DIFFUSION: Wells are punched in agar plate and antiserum and antigen are placed in adjacent wells. Diffusion will occur and precipitin bands will develop between wells containing corresponding antigen and antiserum. The advantage of this method is that there is no necessity to obtain clear antisera and stain extracts.

(3) DOUBLE DIFFUSION IN AGAR GEL: A piece of stained material or extract is placed in a central well cut in agar gel, while each of six wells surrounding this contain a drop of antiserum specific for the globulin or a particular species of animal. The advantage of this method is that the extract can be tested against different antisera simultaneously.

Medicolegal Aspects of Blood Groups: The application of blood grouping to medicolegal problems is based on the following principles: (1) **A blood group antigen cannot appear in a child unless present in one or other parent.** (2) If an

individual is homozygous for a blood group factor, it must appear in the blood of all his children. (3) If a child is homozygous for a factor, the gene for the same must have been inherited by it from each of its parents. (4) **The blood group characters are specific to the individual and are unchanged throughout life.**

EXCLUSION OF PATERNITY: (1) First-order exclusion: Where the child has a blood group gene that is absent in both the mother and the putative father. (2) Second-order exclusion: Where the putative father is homozygous for a blood group gene, but the gene is not present in the child in question.

Agglutinogens: The red cell antigens, representing over 400 serologically determined specificities, belong to a large number of blood group genetic systems with individual chromosomal loci. They are situated in the outer envelope of the red blood cells. They appear in the cells early in foetal life. They are also called isoagglutinogens or antigens (Table 19-1).

Agglutinins: Isoagglutinins appear in the latter half of antenatal life. Agglutinins at birth may be derived from the mother by filtration through the placenta. They diminish or disappear during the first ten days of life, after which the infant produces its own agglutinins.

Blood Groups: The presence of blood group substances is shown by a clumping (agglutination) of the red cells when they are mixed with appropriate antisera. The antisera are prepared from human blood which contain agglutinating bodies called antibodies, which will detect the antigens on the red cells.

The genes which are present in the nuclei of all cells of the body control the formation of the antigens. On Mendelian principle, a person inherits one gene for each blood group from each parent, who themselves may be homozygous or heterozygous for the particular group. If the genes contributed by each parent are the same, the individual is called homozygous for the particular gene and if different, he is heterozygous.

The blood group systems in use are: (1) Red cell antigens. (2) White cell antigens. (3) Serum protein polymorphisms. (4) Red cell enzyme polymorphisms.

(I) RED CELL ANTIGENS

(1) The ABO System: Human blood may be divided into four distinct blood groups, A, B, AB and O, depending upon the attachment to cell membrane of red cells of two agglutinogens which are designated by the letters A and B. The A, B and O, characters are inherited by means of three allelomorphic genes, every individual having two chromosomes each carrying A, B or O, one from each parent. Thus, the possible genotypes are AA, AO, BB, BO, AB, and OO. Group A type may thus be AA or AO. Since there is no true O antigen, group O individuals would be more definitely identified as group H individuals. H-h is inherited independently of the ABO system. The rare hh individuals (Bombay phenotype) lack A, B or H antigens on their erythrocytes and in ABH substances in most secretions. Antigens of the ABO system can be detected even before birth. Their strength goes on increasing after birth till about 3 years of age.

A and B are both "dominant" to O, and O is "recessive" to A and B, whereas A and B are equally dominant. AA, the homozygote, cannot be serologically differentiated from AO, the heterozygote, and the same occurs with the genotypes, BB and BO, the serologically demonstrable blood groups (phenotype) in each case being A and B. The other phenotypes are AB and O. There is no positive method of identifying the "silent" gene O.

ABO group is not found in CSF. Group O is universal donor, and AB universal recipient.

(2) The MNSs System: Two further agglutinogens M and N, which are quite distinct and independent of the agglutinogens A and B, occur in human blood. The M and N factors are inherited as Mendelian dominants. They are present at birth. They form three groups, M, N and MN. Anti-M and Anti-N agglutinins are not normally developed in human sera, but they may be present rarely. The agglutinogens, M and N are feebly antigenic. The subgroup S is closely allied to the MN system. The phenotypes are: MS, NS, MNS, MNs, NSs, MNSs.

(3) The P System: P factor is present in about 75% of persons. It is weak and medicolegally unimportant.

(4) The Rh System: This factor is present in the red cells of 90% of individuals, who are known as Rh-positive, the remainder Rh-negative. A complex of antigens is involved, six in number, named Cc, Dd, and Ee, each of which is capable of producing antibodies, although they are all not equally powerful in this respect. The Rh antigens can be detected in a foetus after 6 weeks of pregnancy.

A member of each pair occupies one of three closely related genes in the relevant chromosome. Eight combinations are therefore possible, and 36 genotypes could result from them. The antibodies do not occur naturally in human serum. They are produced by the entry of a Rhesus antigen into blood, which is foreign to the individual, either due to blood transfusion or from a foetus in pregnancy.

The agglutinogens in the stain are retained for a longer time, even though the red cells are destroyed. The absorption-inhibition technique and absorption-elution method are employed for the grouping of such stains. By using mixed agglutination technique, stain on clothes or fibres can be grouped in about 3-year old stains.

Electrophoresis and immunoelectrophoresis can positively identify blood stains. Separation and migration of haemoglobin and its derivatives can be done by chromatography.

GROUP SPECIFIC SUBSTANCES: The agglutinogens of the ABO system are also present in body tissues. In the tissues, they appear in a lipoidal form. In about 80% of the people, they appear in a water-soluble form and can be demonstrated in all the body fluids except the cerebrospinal fluid. Persons who possess only the lipoidal form are known as 'non-secretors' while those who possess a water-soluble form are known as 'secretors'. The capacity of secreting these antigens in body fluids is controlled by a pair of allelic genes Se and se, the former being dominant over the latter. The individuals with genotype Se Se and Se se are secretors and those with the genotype se se are non-secretors. Secretors possess H antigen on their red cells irrespective of their blood group of the ABO system. The agglutinins, a and b are also present in the body fluids. **M and N agglutinogens are widely distributed in the body tissues in a relatively water-soluble form.** The group specific substances in dried stains can be identified by absorption technique. The **Rh agglutinogens are widely distributed in the body tissues but are not found in the body fluids, except the amniotic fluid.**

BLOOD GROUPS AND HEREDITY: Bernstein postulated the mode of inheritance of ABO system. The ABO, MN, and Rh factors are inherited according to Mendelian principles. **The rules of inheritance of ABO system are:** (1) Agglutinogen A or B cannot appear in the child unless it is present in one or both parents. (2) Agglutinogen A_1 or A_2 cannot appear in the blood of the child unless it is present in one or both parents. (3) The combination of A_1B parent with A_2 child and vice versa cannot occur. (4) An O parent cannot have an AB child and an AB parent cannot have O child. (5) Parents of AO and AO genotype may have a OO child. (6) Parents of AA or AO genotype may have A child **(Table 19-1)**.

Table (19-1). Inheritance of blood groups

Phenotypes of Parents	Phenotypes of Children	
	Possible	Impossible
OXO	O	A, B, AB
OXA	O, A	B, AB
OXB	O, B	A, AB
OXAB	A, B	O, AB
AXA	O, A	B,AB
AXB	O, A, B, AB	NONE
BXB	O, B	A, AB
BXAB	A, B, AB	O
ABXAB	A, B, AB	O
MXM	M	N, MN
MXMN	M, MN	N
MXN	MN	M, N
MNXMN	M, N, MN	NONE
MNXN	N, MN	M
NXN	N	MMN

The rules of inheritance for MN system are: (1) Agglutinogens M and N, cannot appear in the blood of a child unless present in one or both parents. (2) A type M parent cannot produce a type N child, and conversely an N parent cannot produce M child. (3) In matings where both parents are homozygous type M or N, the children are always of the same type as the parents. (4) In matings where one parent is type M and other type N, all children are type MN. (5) In matings where one parent is homozygous (M or N), the children are of parental types in 50:50 ratio. (6) In matings where the parents are both MN, children of all three types are possible **(Table 19-2)**.

Rules of inheritance of Rh groups are: (1) Rh negative parents cannot produce an Rh-positive child. (2) Rh-positive and mixed parents can have Rh-positive and Rh-negative children.

Blood groups in tissues: Blood groups can be determined from soft tissues, hair, nails, dental tissue and bone.

White Cell Antigens: The Human Leucocyte Antigen (HLA) system consists of protein substances on the surface of a wide variety of tissues and organs, on tumours, white cells and platelets. They are present on the placenta at term and in foetal tissue at 6 weeks. They

Table (19-2). Inheritance of the M-N factors

Group of Parents	Group of Children	
	Possible	Not Possible
MXM	M	N, MN
M XMN	M, MN	N
M XN	MN	M, N
N X N	N	M, MN
N X MN	N, MN	M
MN X MN	M, N, MN	None

are found on both lymphocytes and granulocytes. The major human leucocyte antigens HLA-A, B, C, D, and DR are determined by a single chromosomal segment, the "major histocompatibility complex" (MHC), which is situated on the short arm of human chromosome six. In white cells 68 antigenic factors are present. Each parent contributes to a child, one of the two chromosomes containing the HLA region. The presently known seven multiple loci with about 125 multiple allelism, and with a very high degree of polymorphism, provide a very good discriminating capacity. Only fresh blood samples are examined in solving the problems relating to parentage. In tissue grafts, the better HLA match between subjects, the better the chances of graft's survival. The presence of these antigens may be detected by using the white cells of the blood and a suitable antiserum, that either agglutinates the white cells or damages them, so that they are stainable with suitable dyes that do not affect the damaged cells.

They are usually demonstrated by electrophoresis.

SERUM PROTEIN POLYMORPHISM: The serum protein groups which are subject to genetic variation include the Gm and Inv types of gamma globulin, the group specific components (Gc types), the haptoglobin (Hp) types, the transferrin types (Tfc), and several lipoprotein systems. They are usually demonstrated by electrophoresis.

(1) Serum Haptoglobins: Haptoglobins are haemoglobin-binding proteins found in human serum. Three genotypes are present Hp_{1-1}, Hp_{2-2} and Hp_{2-1}. The pattern of inheritance is identical to that of the MN groups.

(2) Gc Groups: Inheritance is controlled by two alleles Gc 1 and Gc 2 giving the three phenotypes Gc 1-1, Gc 2-2 and Gc 2-1.

(3) Ag Groups: This system of serum antigens is complex but two genes, Ag(x) and Ag(y) appear to be allelic. These factors are present at birth and not transmitted across the placenta.

(4) Gm Blood Serum Polymorphism: In the newborn, the gamma globulin present is not its own, but has been transmitted from the mother across the placenta. As such, Gm and Inv. studies are useful in cases of mix up or exchange of babies in hospitals. The child's own gamma globulin begins to form from third month with disappearance of the Gm transferred from the mother. There are five types of immunoglobulins, IgG, IgM, IgA, IgD and IgE, which form a group of serum proteins to which all antibodies belong. Normal serum contains eight types of IgG molecules. Various Gm antigens designated Gm 1 to Gm 25 are so far determined by various allelic genes.

(5) Km System: At the Km locus, there appear to be genes giving rise to four allotypes known as Km1, Km2, Km3, and Km Blank.

(6) Serum Lipoproteins: The Lp system has phenotypes Lp (a+x–), Lp (a+x+) and Lp (a–x–).

(7) Abnormal Haemoglobins: The abnormal haemoglobins are under direct genetic control. More than hundred variants of haemoglobin have been described. The specific mutation is known for at least 30 Hb variants.

RED CELL ENZYME POLYMORPHISMS: More than 160 antigens, 150 serum proteins and 250 cellular enzymes have been found in the red cells. A large number of enzymes, which catalyse the various vital biochemical reactions, are also present in the blood, mainly in the plasma. Phosphoglucomutase (PGM) and adenylatekinase (AK) are of established value in paternity testing. Adenosinedeaminase (ADA), red cell acid phosphatase (EAP), serum cholinesterase (SCE), 6-phospho-gluconate dehydrogenase (6-PGD), glutamate pyruvate transaminase (GPT), esterase (EsD) and glyoxalase 1 (GLO), may also be used. They are usually demonstrated by the starch gel electrophoresis technique.

Paternity can be excluded by ABO grouping alone in about 18%, and if MNSs system is added in 44%, and if Rh groups are added in about 60%. Red cell antigens and serum proteins exclude 82%; if red cell enzymes are included exclusion will be 94%. By testing a number of HLA systems, the exclusion is 98.5%.

Medicolegal Application of Blood Groups:

(1) Disputed Paternity: The question of disputed paternity arises in the Court in the following conditions: (1) When a child is born in lawful marriage, but the husband denies that he is the father of the child. (2) When a child is born out of lawful marriage and the mother accuses a certain man of

being the father of the child, while the man denies the accusation. (3) When a woman pretends pregnancy and delivery and obtains a child claiming it as her own, in order to obtain a share in her husband's property. (4) In suits for nullity of marriage.

Many cases can be solved by means of the blood groups of the parent and the child. In the case of adults, 5 ml. of venous blood is taken and placed in plain tube. Neither party should have had a blood transfusion within three months before taking the sample. The infant should preferably be at least six months of age, but not less than two months before testing is performed. One millilitre of blood should be obtained by a heel or ear prick, or venepuncture into a plain tube. Mutation of the genes (non-inherited germline) in a child can give rise to wrong results in paternity testing.

The tests have their limitations. They may exclude a certain person as the possible father of the child, but they cannot definitely establish paternity. They can only indicate its possibilities. The blood groups in current use in the investigation of cases of doubtful paternity are ABO, MNS, Rh, Kell, Lutheran, Duffy and Kidd.

(2) Disputed Maternity: When the same child is claimed by two women or when two children are interchanged either by accident or by design in maternity home or hospital, or supposititious child and when a woman kidnapping a child and claiming to be the mother of that child, blood grouping tests are helpful.

(3) Crimes: (1) Blood stains may be found on clothing and person of suspect. If the accused alleges that the stain is of his own blood, it will have similar blood group systems and haptoglobins. If the victim has similar characters, the test is not conclusive. If there is difference in blood group of the stain and the accused's blood, then the stain is of some other person's blood. If the characteristics of the victim's blood are similar with those of the stain, an association is established between the suspect and the victim. (2) Blood stains may be present at the scene of house-breaking, e.g., on a broken window, if the thief has cut himself. If the character of these stains is similar to that of blood of the suspect, it establishes association. (3) Blood stains may be present under the fingernails of assailant in a case of throttling. If there has been a struggle, blood stains derived from the accused may be found under the fingernails of the victim, due to scratching. (4) Vehicles which have caused injury can be identified when they show blood resembling that of the victim.

Stains on clothes due to crushing of bugs, fleas, louse, mosquitoes, etc., are common. These stains are small in size and sharply angular in outline and are usually found on the inside of the garment. If the insects are crushed, fragments of the hair or scales of the insect and eggs may be found on microscopic examination.

(4) Stains due to Body Fluids: The blood group agglutinogens can be demonstrated in stains on clothes due to semen, sweat, saliva, nasal secretion, urine or faeces in persons who are "secretors". This may be a corroborative evidence of the accused.

(5) Identity: The specificity of various blood group combinations is like that of the fingerprints. When an individual has some rare blood group, he can be identified with certainty. But when they are of common type, they are not of use.

(6) Cause of Death: In certain cases cause of death can be established, e.g., incompatible blood transfusion. Poisons can be detected in the blood.

DNA FINGERPRINTING

Each human nucleus contains about a metre of DNA, but only 5% is used for genetic coding, the rest being redundant or silent segments [stutters; hypervariable regions (HVR); minisatellites]. Of these redundant segments, there may be 200 to 14,000 repeats of each identical sequence on each DNA strand. These segments of nucleotides are called "repetitive DNA". The length, constitution, and number of the repetitive sequences are different for each person, but are unique for an individual (except uniovular twins), and are stably inherited in a Mendelian fashion. This method is as unique as fingerprints to an individual.

(1) RFLP Method (Restriction Fragment Length Polymorphism): Nucleated cells are the source of DNA for extraction from blood, semen, vaginal epithelial cells, tooth pulp, bone marrow, hair roots, muscle, skin, mucous membranes, etc. A sample is taken and from it the DNA is chemically extracted and purified. It is then cut into fragments at specific

base sequences by a restriction enzyme (RE). When this process is repeated by several enzymes, each of which cuts at different sites, enough information is gathered to construct a detailed genetic fingerprint of a person. Because every person's DNA sequence is different, the fragments in the DNA specimen from one individual to another are different in number and length, from those in a DNA specimen from another individual. Next, the double stranded DNA are denatured into single strands. The DNA fragments are then separated by gel electrophoresis that spreads the fragments into bands. DNA carries a negative charge and thus travels towards the positive pole in electrophoresis. The distance that each fragment travels depends on its length. The longer the fragment, the slower its rate of migration. A number of DNA bands are produced in the gel. These are transferred to nylon or nitrocellulose sheets by Southern blotting technique. This process fixes the DNA fragments to the membrane firmly and in the same position.

The membrane is then exposed to a DNA probe, which is a single strand of DNA labelled with radioactive phosphorus-32, which specifically binds to the core sequences. X-ray film is put in direct contact with probe-labelled membrane to detect the radioactive patterns, which appear as a series of 30 to 40 parallel bands or bars. Each dark band represents a point where the DNA probe is bound to its complementary base sequences. From the presence of different bars in given positions, comparisons can be made with other samples. It is possible to identify a person from a single human cell. The chance of two persons sharing the same sequence is about one in thirty thousand billions. Rarely, there are mutations confined to one band.

(2) POLYMERASE CHAIN REACTION (PCR): This technique is used when a very small amount of DNA or a partially degraded biological material is available. It can analyse 36 samples at a time. It takes into account polymorphic DNA segments present in the specimen, and by polymerase chain reaction (PCR) produces multiple copies of a particular polymorphic DNA segment, sufficient to meet the requirements of DNA profiling test. The advantages of PCR over the RFLP are: (1) It is relatively simple and easily carried out in the laboratory. (2) The results are obtained within few days. (3) It permits analysis of extremely tiny amounts of DNA. Application of polymerase chain reaction has revolutionised the field of applied forensic medicine.

PCR is now widely being used by all laboratories, as it is very sensitive, reaction is highly specific, easily automated and capable of ampliflying minute amount of samples, even if the biological material is degraded.

(3) SHORT TANDEM REPEATS (STR) ANALYSIS: It uses highly polymorphic regions that have short repeat sequences of DNA. The most common is 4 bases repeats, but there are 3 and 5 bases repeats in use. Because different people have different numbers of repeat units, these regions of DNA can be used to discriminate between individuals. This technique can be done in 2 to 3 days. It can be performed on small quantities of DNA.

(4) Amplified Fragment Length Polymorphism (AmpFLP).

(5) Mitochondrial DNA Analysis: (mt DNA): While older biological samples that lack nucleated cellular material, such as hair, bones and teeth cannot be analysed with STR and RFLP, it can be analysed with mt DNA. In the investigation of cases that have gone unsolved for many years, mt DNA is extremely valuable.

(6) Y-Chromosome Analysis: It is especially useful for tracing relationships among males, or for analysing biological evidence involving multiple male contributors.

Medicolegal Importance: (1) The blood on a weapon can be matched against the blood of the victim. (2) Hair roots found on a weapon can be matched against the blood of the victim and accused. (3) Seminal fluid recovered from the vagina of a victim can be matched against the blood of an accused. (4) Disputed maternity. (5) Exchange of new born in hospitals. (6) It can exonerate a falsely implicated person in a crime. (7) Paternity can be established positively. (8) It can be applied for tracing pedigrees and for establishing family relationship. (9) Identification of victims of accident, mass disasters, and mutilated bodies can be made by matching prints with prints of parents or close relatives.

Hazards of blood transfusion: (A) Immunological reactions. (1) Intravascular haemolysis. (2) Extravascular haemolysis. (3) Sensitivity to white blood cells, platelets and plasma components. **(B) Non-immunological reactions:** (1) Circulatory overload. (2) Coagulation defect. (3) Hyperkalaemia. (4)

Citrate toxicity. (5) Infections, serum hepatitis, AIDS, etc. (6) Air embolism. (7) Hypothermia.

VARIOUS STAINS: SALIVA: It contains enzymes like ptyalin, 6-phosphogluconate dehydrogenase, various proteins, lipids, chlorides, thiocyanate ions, etc. The stains are identified from the presence of amylase and buccal epithelial cells. ABO grouping and species origin can be carried out.

FAECES: The stains can be identified from odour, presence of undigested muscle and vegetable fibres and stercobilin.

VAGINAL SECRETION: It consists of white coagulated material consisting of shed vaginal epithelium and Doderlein's bacilli.

VOMITUS: It will contain food material and reaction is acid.

PLATE 1

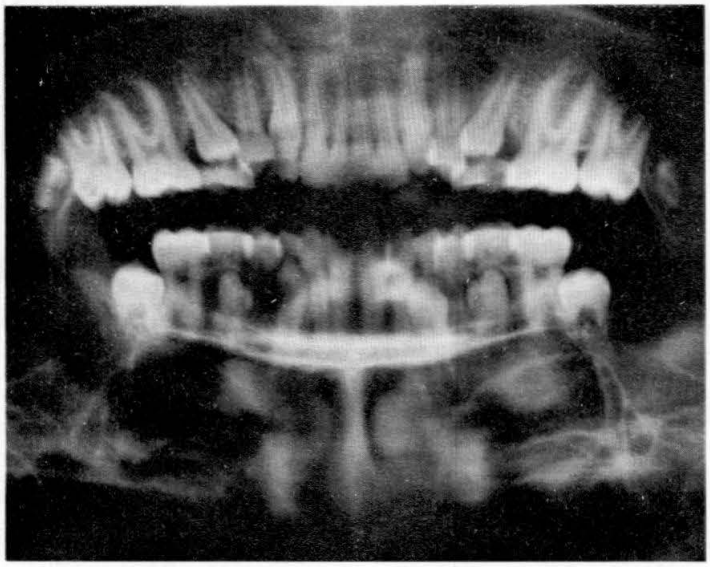


Fig. 1. **Orthopantogram showing mixed dentition is useful to determine age.**

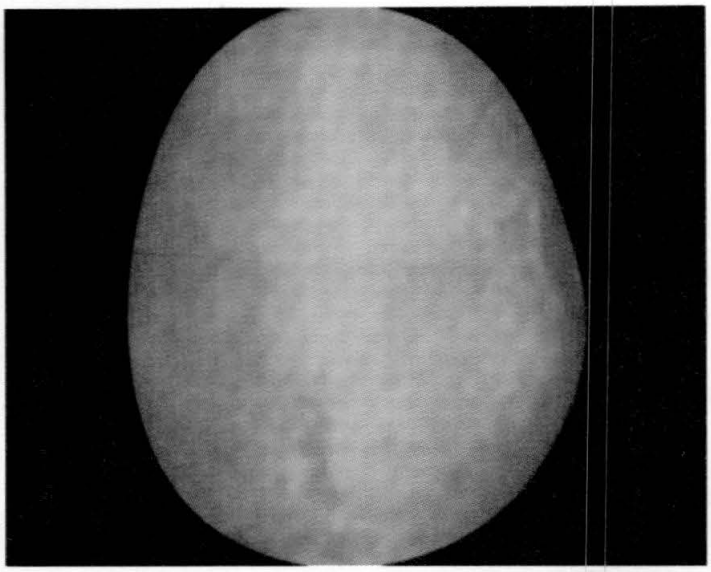


Fig. 2. **Complete fusion of the sutures of the skull.**

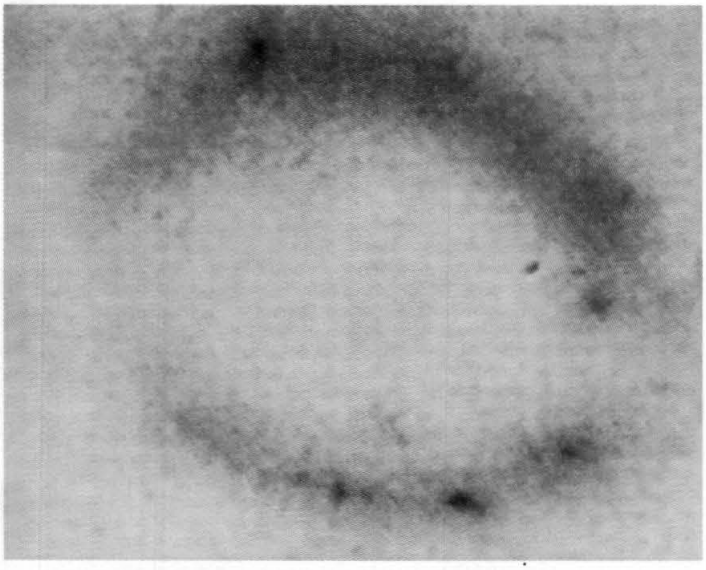


Fig. 3. **Bite mark.**

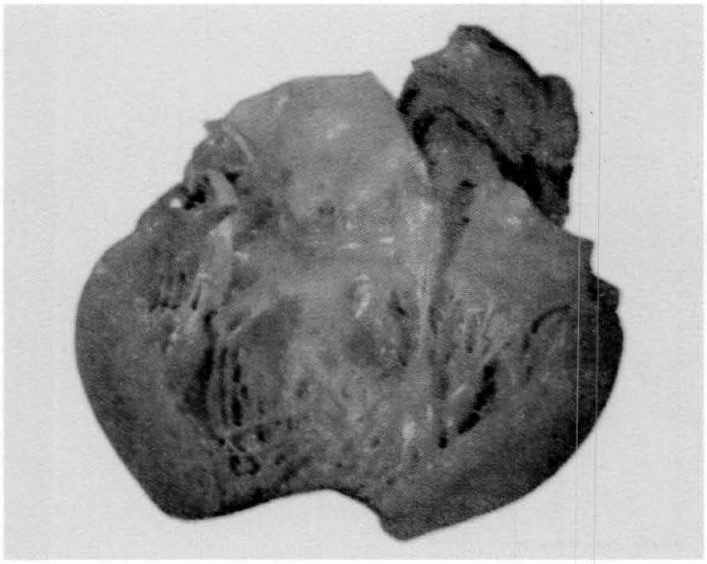


Fig. 4. **Subendocardial haemorrhages on the interventricular septum.**

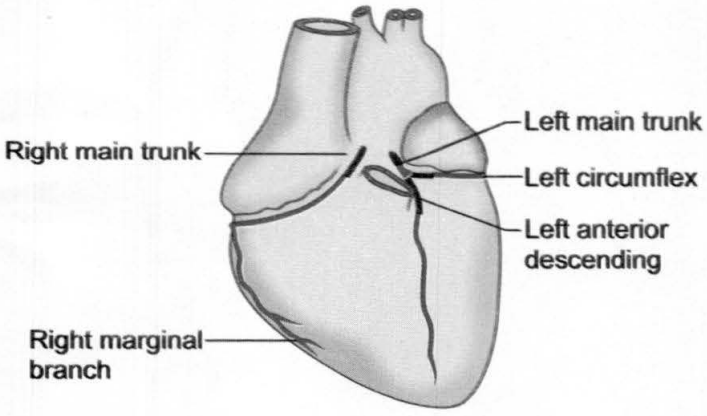


Fig. 5. **The main coronary arteries with the most frequent sites of occlusion.**

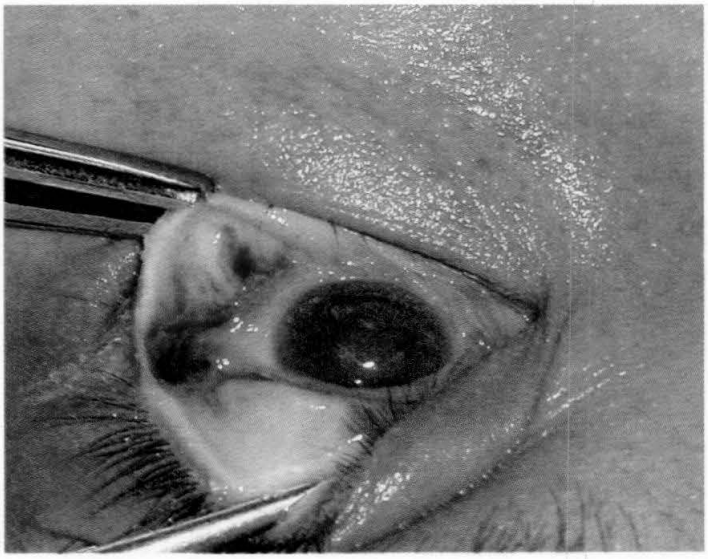


Fig. 6. **Subconjunctival haemorrhages.**

PLATE 2

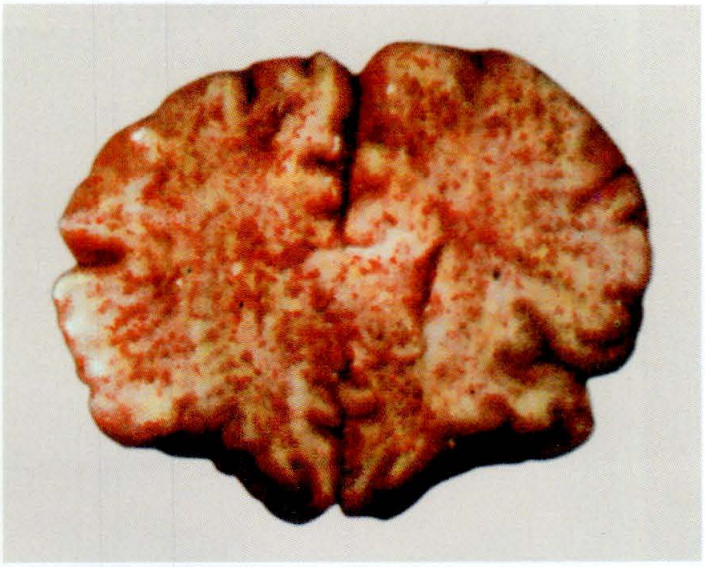


Fig. 7. Petechial haemorrhages in the brain.

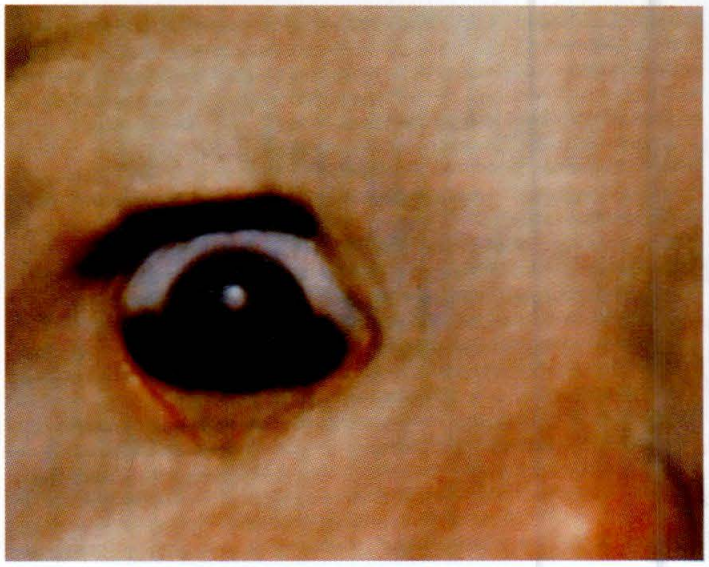


Fig. 8. Tache noir.

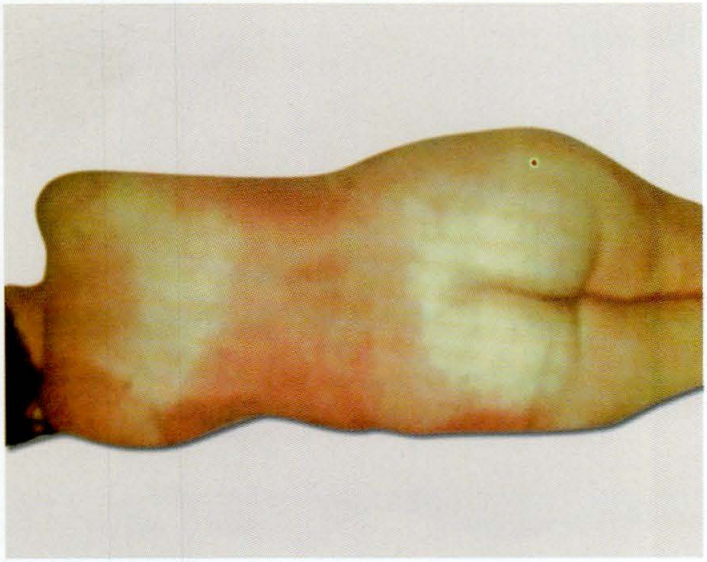


Fig. 9. Postmortem hypostasis, and areas of contact flattening.

Fig. 10. An incision into hypostatic area showing congestion.

Fig. 11. Cherry-red PM hypostasis in CO poisoning.

Fig. 12. Chocolate-coloured PM lividity in sodium chlorate poisoning.

PLATE 3

Fig. 13. Greenish discolouration of right iliac fossa.

Fig. 14. Gaseous distension of body due to decomposition.

Fig. 15. Advanced decomposition. Marbling, bloating and skin peeling are seen.

Fig. 16. Blackening of body and maggots in advanced decomposition.

Fig. 17. Early stage of adipocere formation.

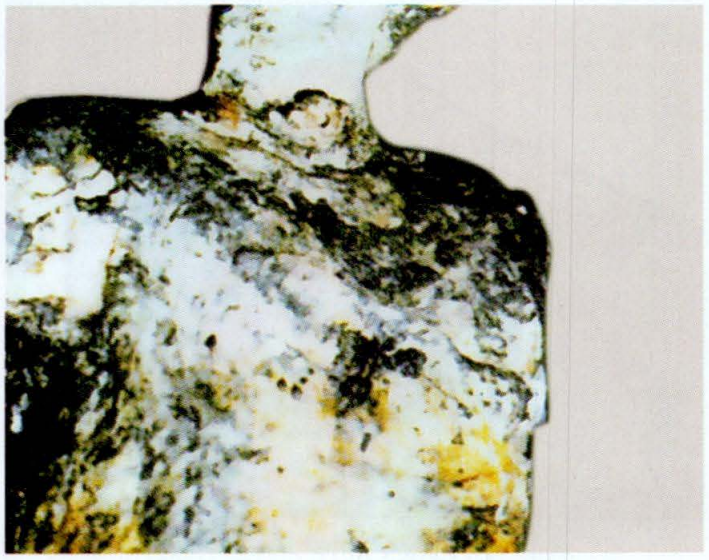


Fig. 18. Adipocere.

PLATE 4

Fig. 19. Abrasion of left knee.

Fig. 20. Multiple small abrasions on forearm.

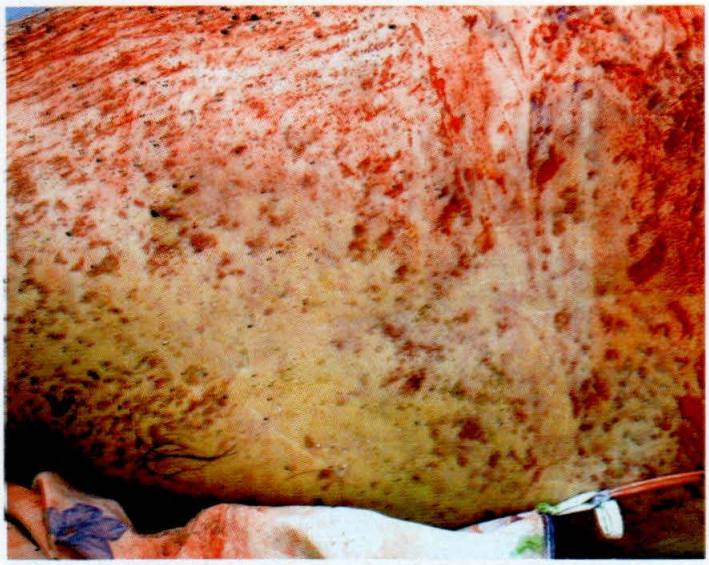


Fig. 21. Gravel abrasions due to fall from a height.

Fig. 22. Grazed abrasion over face.

Fig. 23. Brush burn on the back.

Fig. 24. Injury: Graze abrasion patterned.

Fig. 25. Patterned abrasions caused by tyre tread.

Fig. 26. Multiple bruises on the forearm.

Fig. 27. Bruising of the eye.

Fig. 28. Multiple patterned contusions on lower extremities caused by a stick on both lower limbs.

Fig. 29. Patterned contusions caused by a stick.

Fig. 30. Patterned contusions over buttock.

Fig. 31. An incision to demonstrate deep contusion in the calf muscles.

Fig. 32. An incision to demonstrate deep contusion on the back of the hand.

Fig. 33. Artificial bruises caused by application of juice of marking nut.

Fig. 34. Lacerated wound on knees.

Fig. 35. Laceration of scalp.

Fig. 36. Avulsed laceration of scalp.

Fig. 37. Multiple punctate lacerations caused by broken glass pieces.

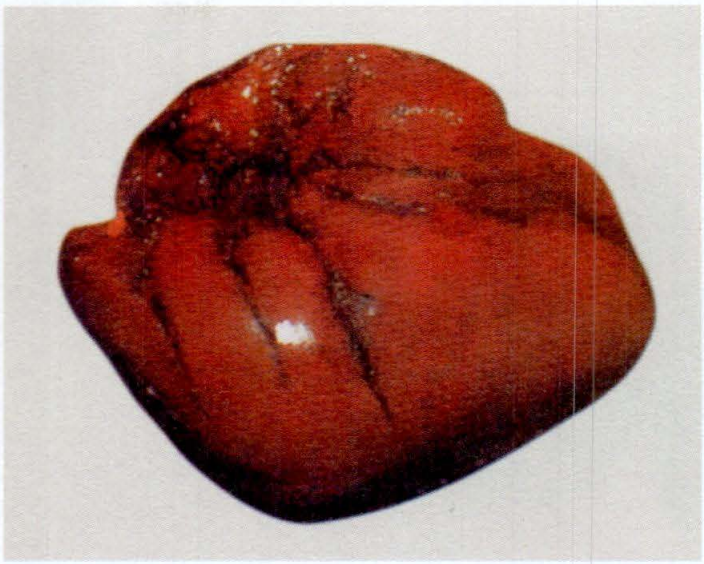


Fig. 38. Multiple lacerations of the liver.

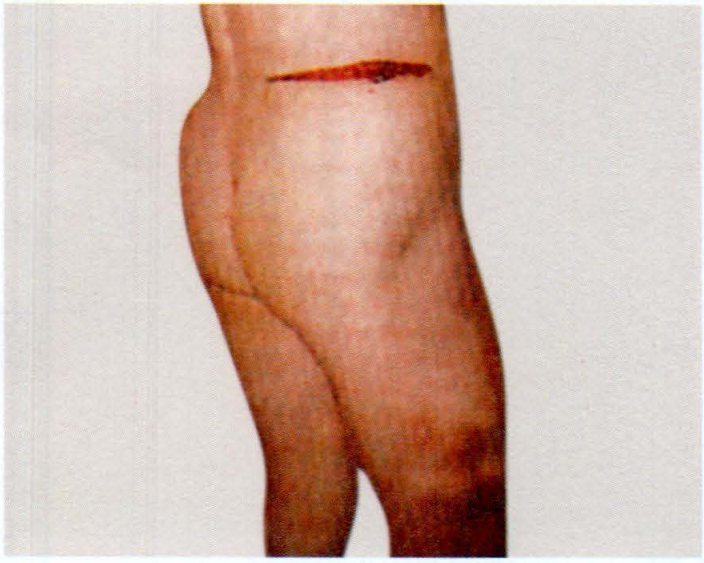


Fig. 39. Incised wound on the flank.

Fig. 40. Suicidal incised wound on the front of the wrist.

Fig. 41. Self-inflicted superficial incised wounds.

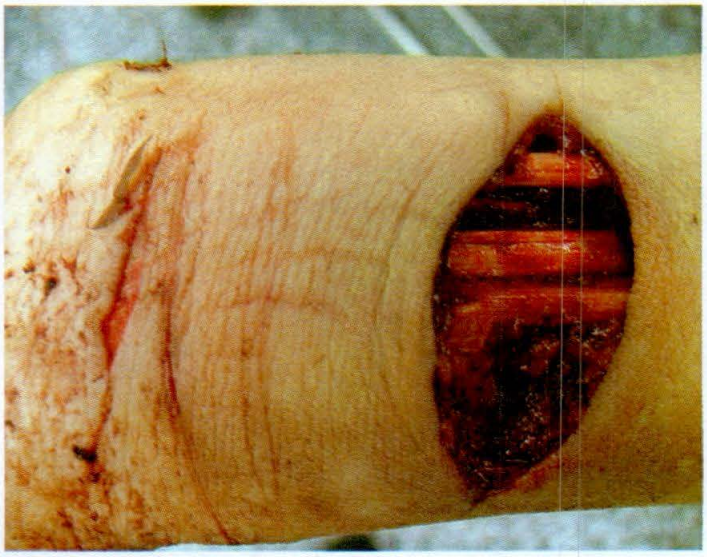


Fig. 42. Multiple hesitation cuts and a gaping incised wound.

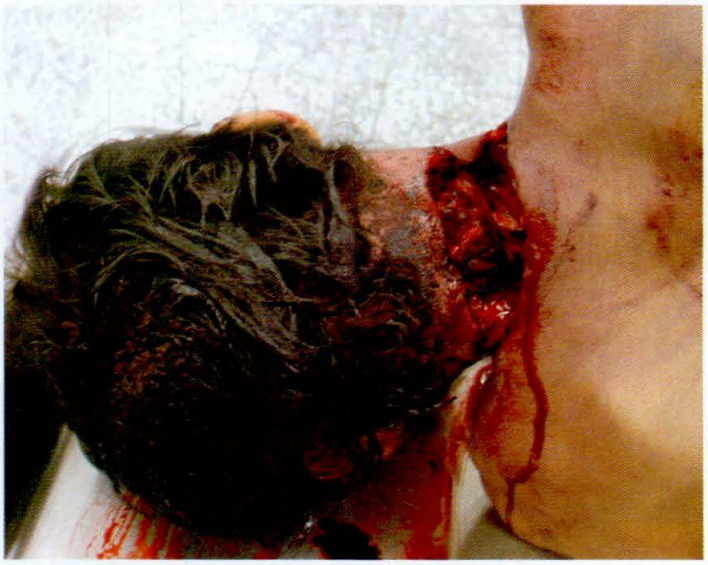


Fig. 43. Chop wound on back of neck.

Fig. 44. Decapitation and above a chop wound.

Fig. 45. Chopped fingers of hand.

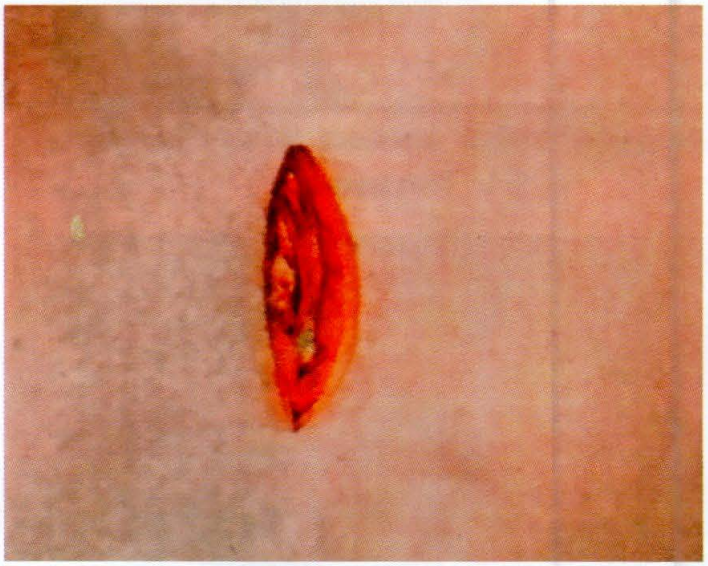


Fig. 46. Stab wound with a single-edged weapon.

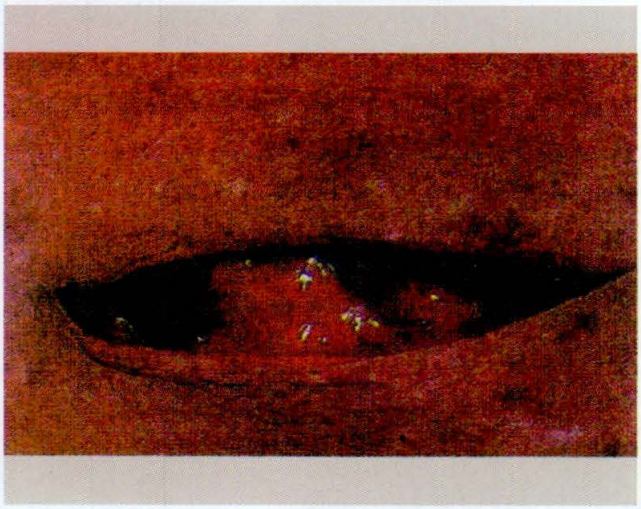


Fig. 47. Stab wound with a single-edged weapon.

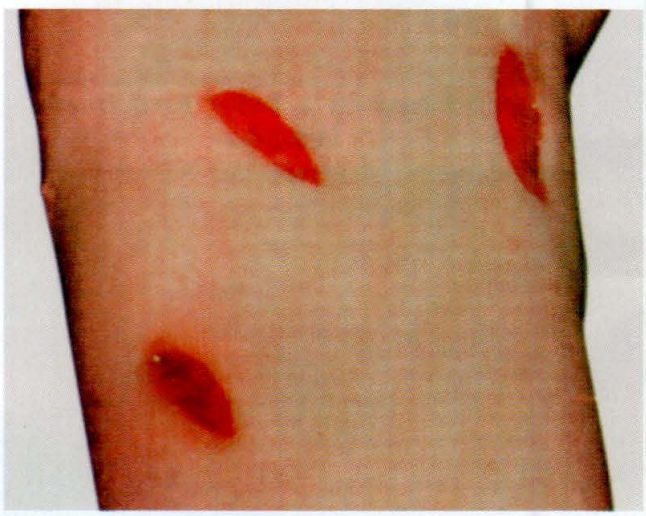


Fig. 48. Multiple stab wounds.

PLATE 9

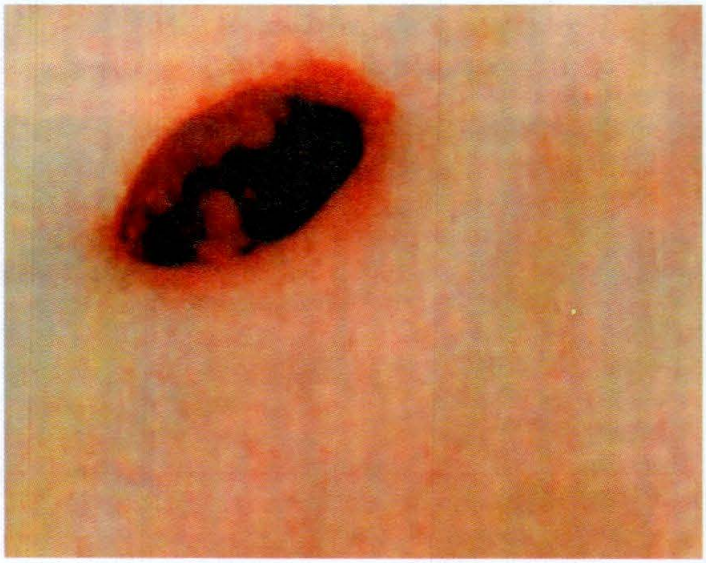


Fig. 49. Stab wound showing gaping.

Fig. 50. Multiple stab wounds some of them showing tailing.

Fig. 51. Stab wound of heart.

Fig. 52. Shotgun wounds from a distance of about 10 metres.

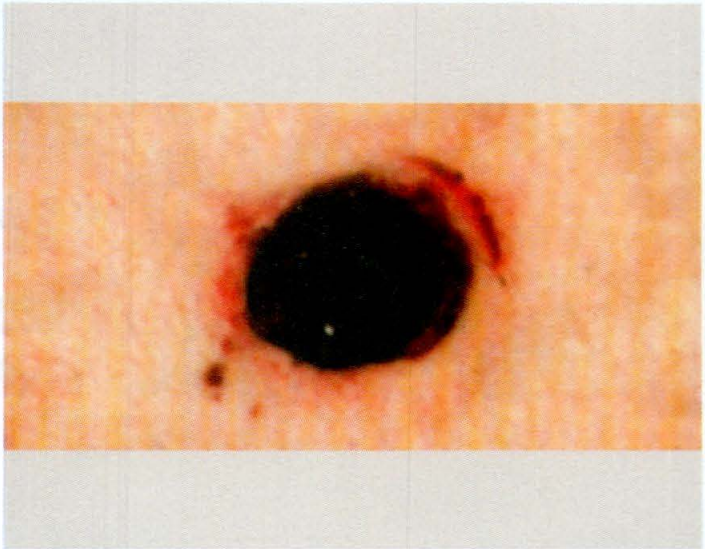


Fig. 53. Tight-contact shotgun wound of chest. Muzzle imprint is seen on the upper right side.

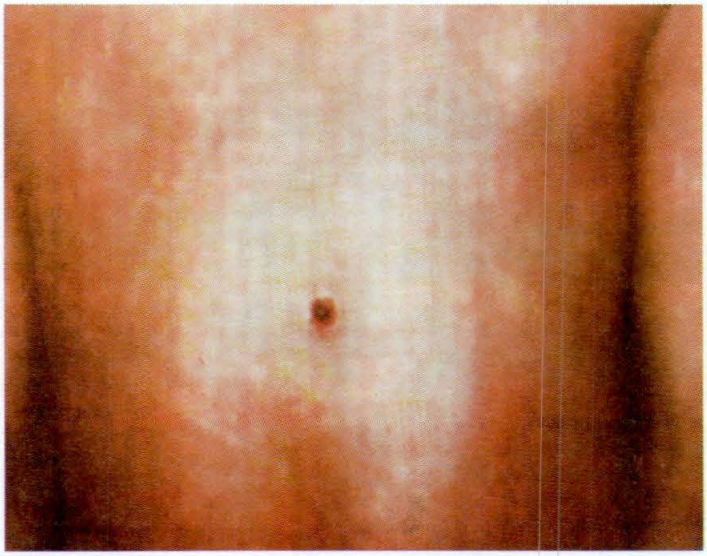


Fig. 54. Distant shotgun wound caused by a rifle slug.

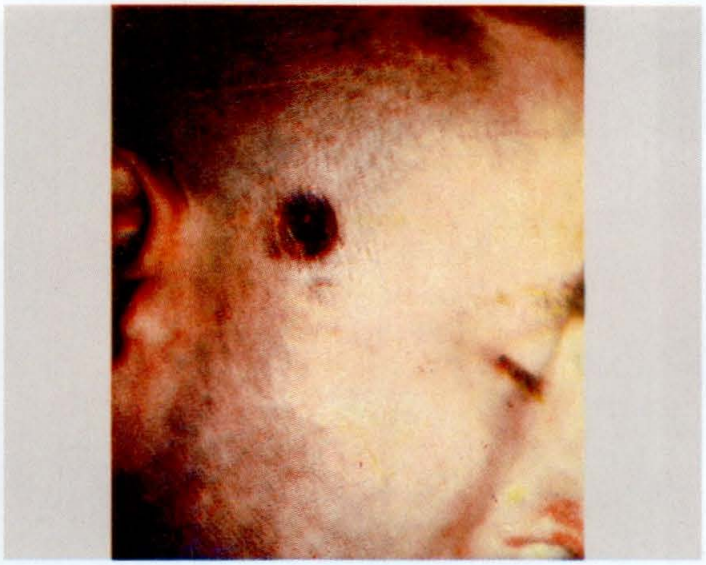


Fig. 55. Suicidal contact wound by revolver in the temple.

Fig. 56. Bullet wound of close range.

Fig. 57. Near entry wound from a revolver.

Fig. 58. Near contact entry wound from a revolver.

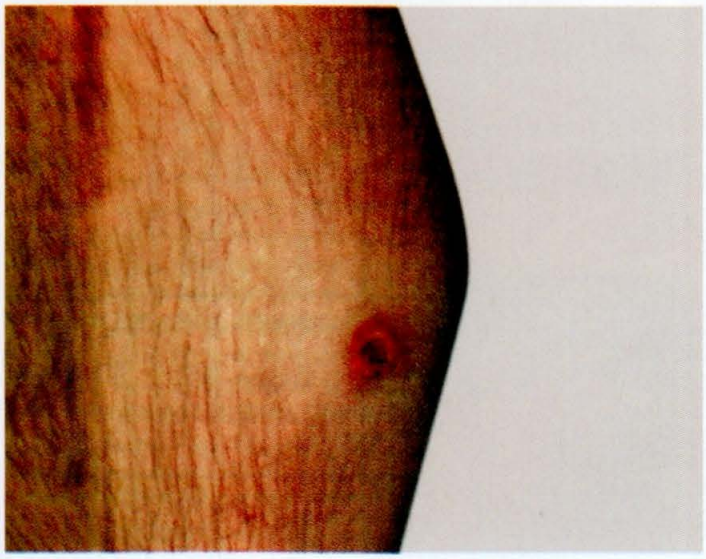


Fig. 59. Entry wound by a bullet fired from a distance.

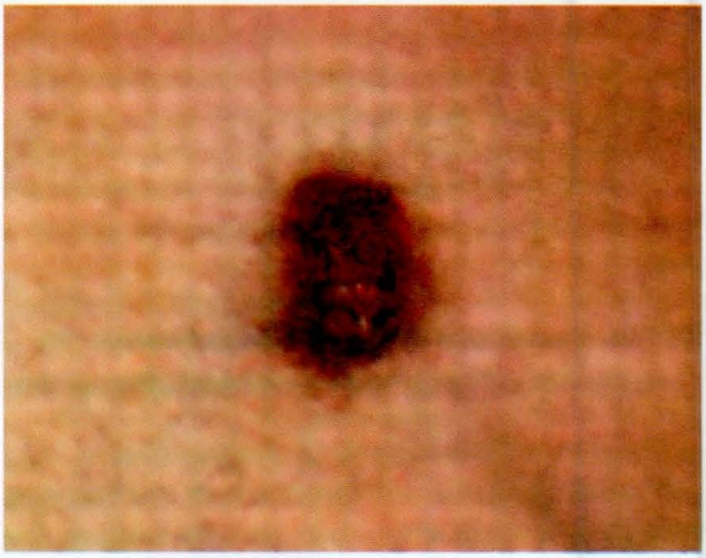


Fig. 60. Distant bullet wound showing abraded skin below the entrance.

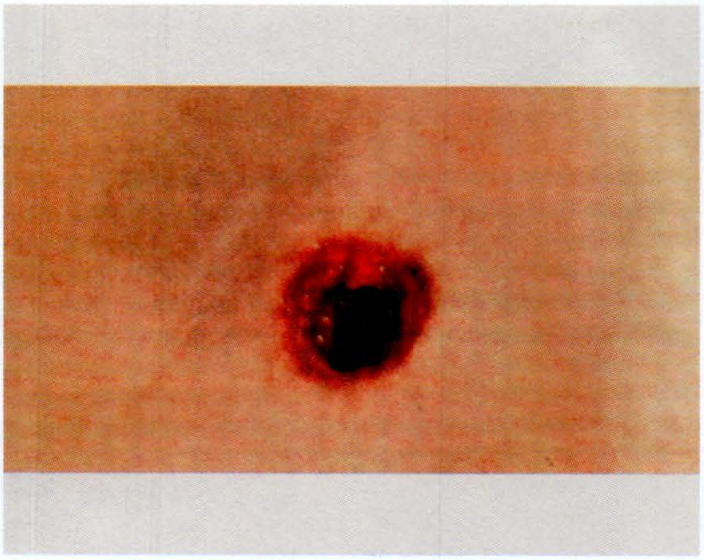


Fig. 61. Shored exit wound.

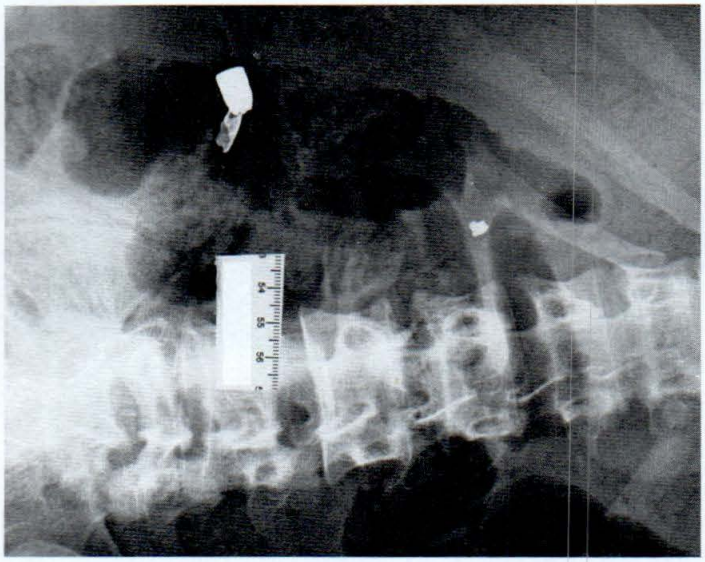


Fig. 62. X-ray photograph showing a fired bullet in the body.

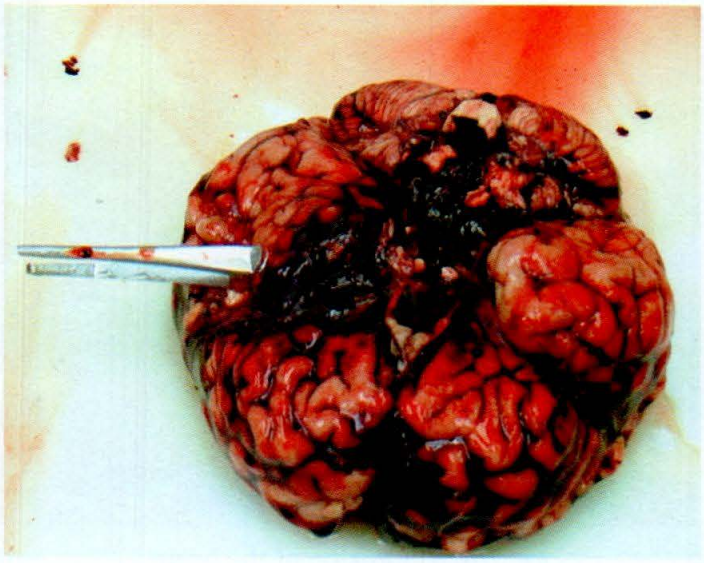


Fig. 63. Track and cavity caused by bullet passing through the brain.

Fig. 64. Defence wounds on shoulder and upper arm.

Fig. 65. Defence wounds on fingers and wrist.

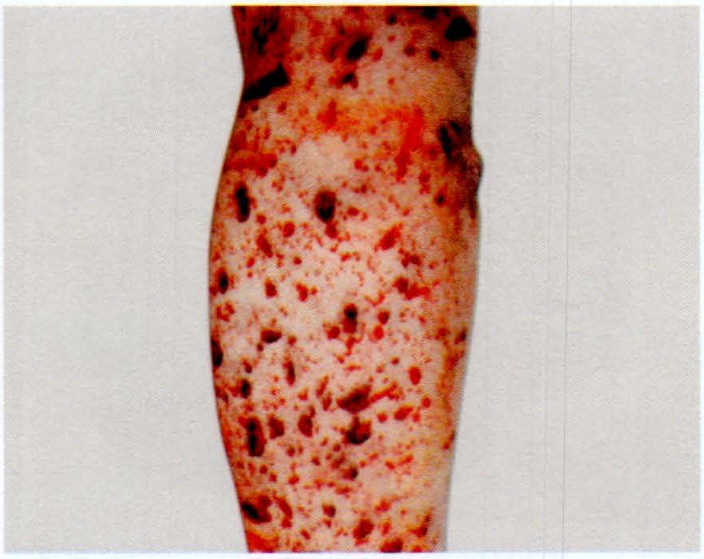


Fig. 66. Bomb explosion showing triad of abrasions, contusions and lacerations.

Fig. 67. Injuries caused due to run over by train.

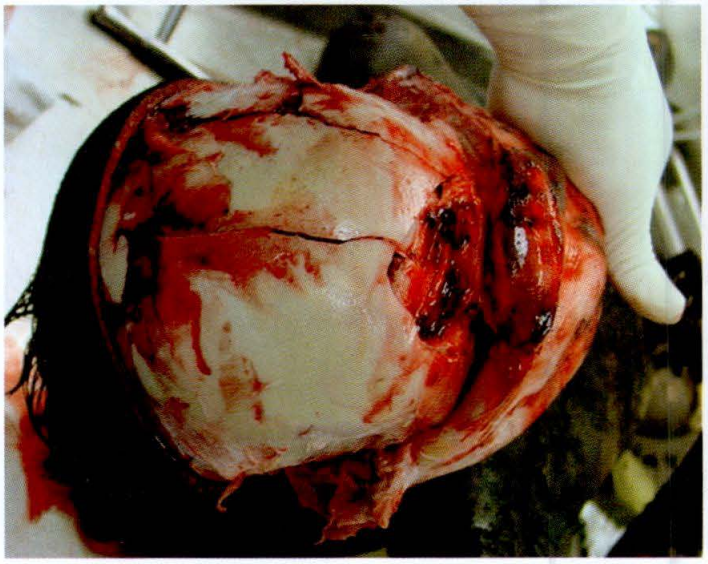


Fig. 68. Depressed and fissured fracture of the skull.

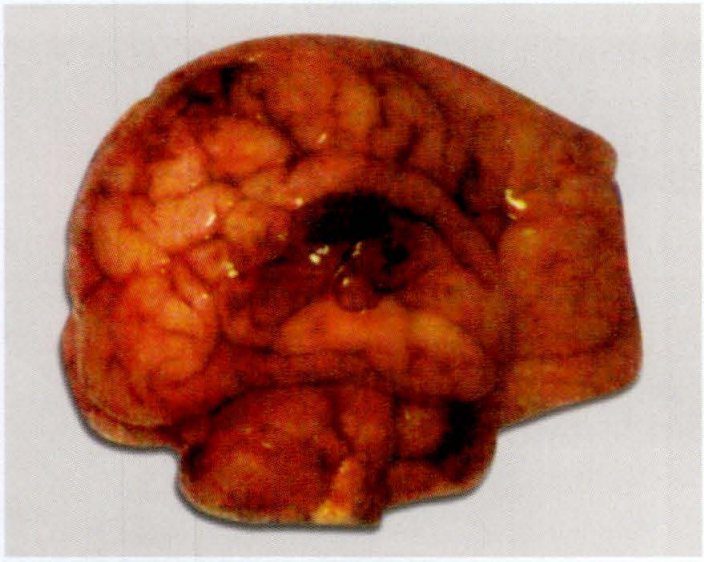


Fig. 69. Contusion of the right lateral side of the brain with subarachnoid haemorrhage.

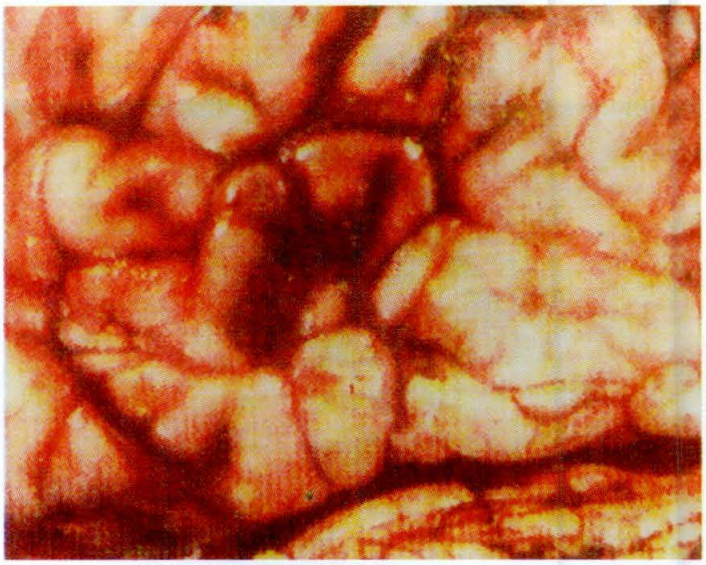


Fig. 70. Contusion of the brain.

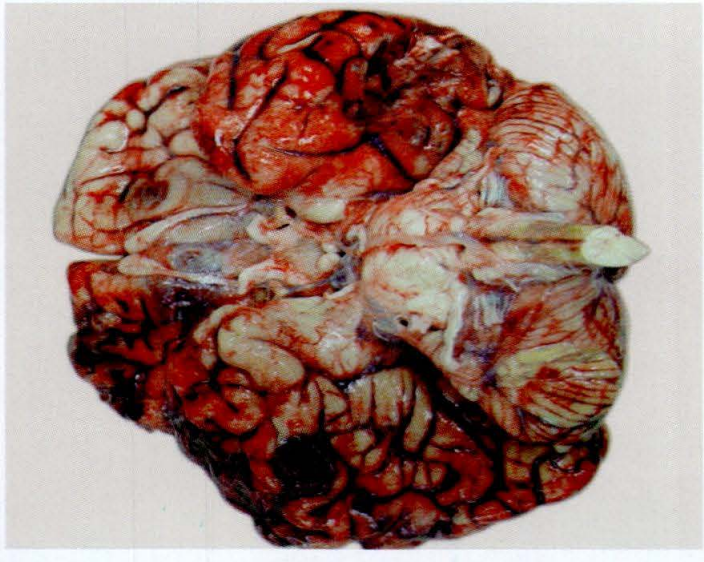


Fig. 71. Contusion of the lower surface of the brain.

Fig. 72. Contusion and lacerations of base of the brain.

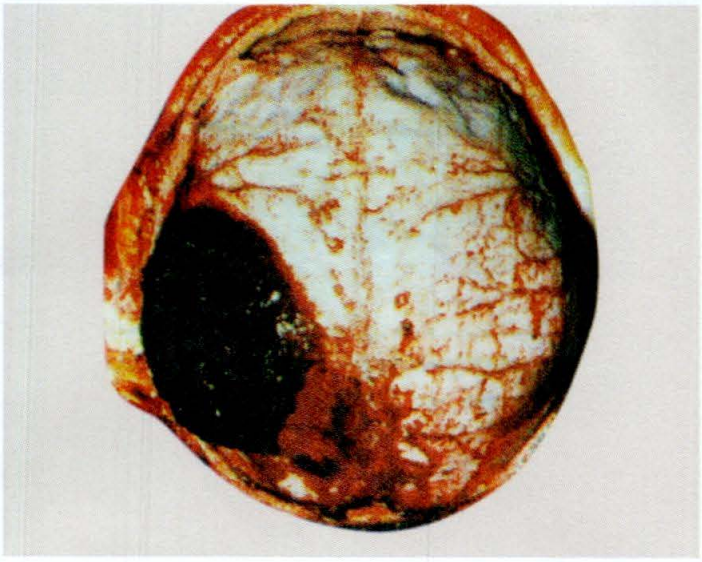


Fig. 73. Extradural haemorrhage.

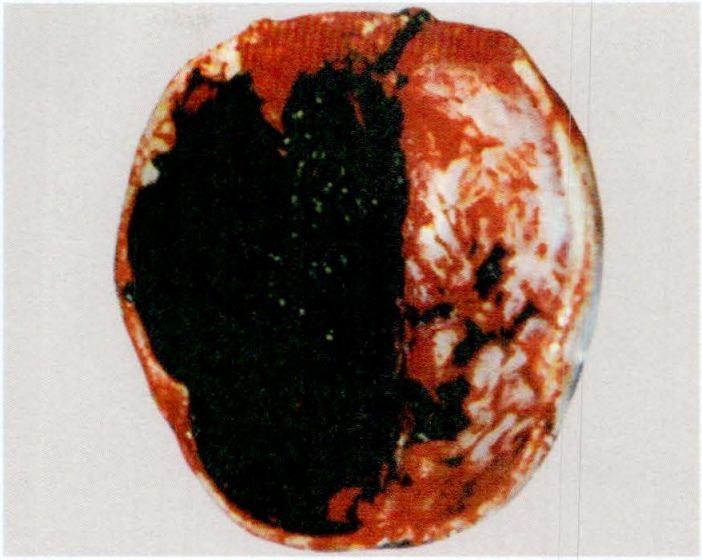


Fig. 74. Subdural haemorrhage on base of the skull.

Fig. 75. Subdural haemorrhage.

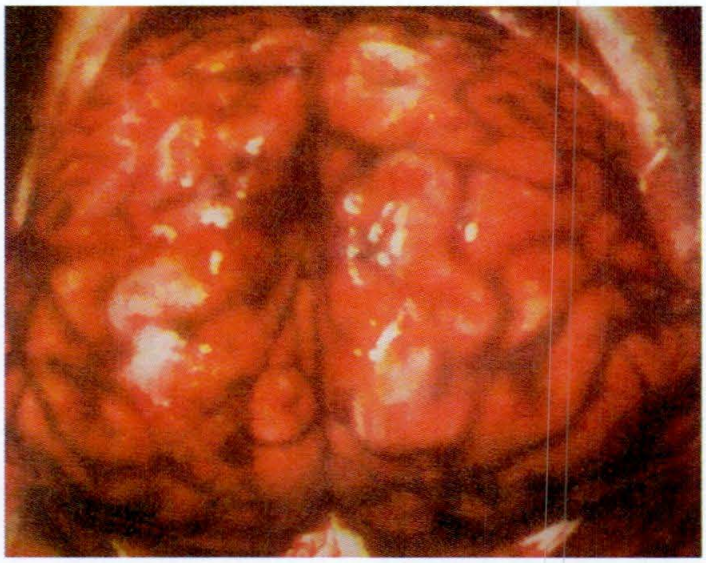


Fig. 76. Subarachnoid haemorrhage.

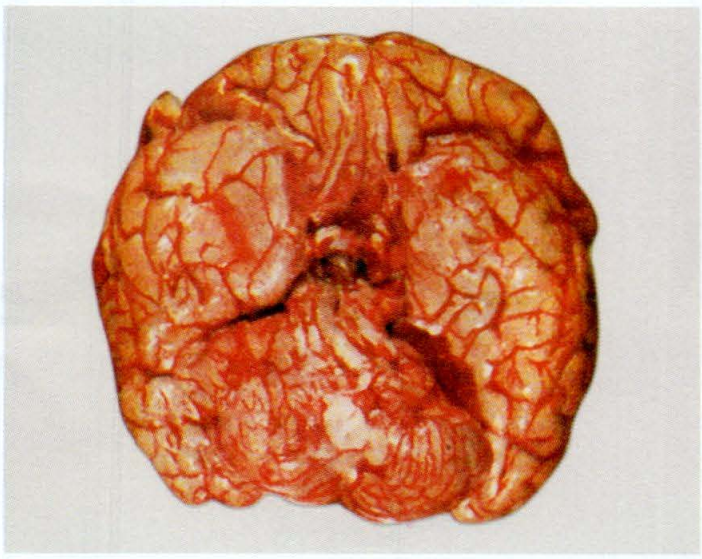


Fig. 77. Large berry aneurysms of posterior communicating artery.

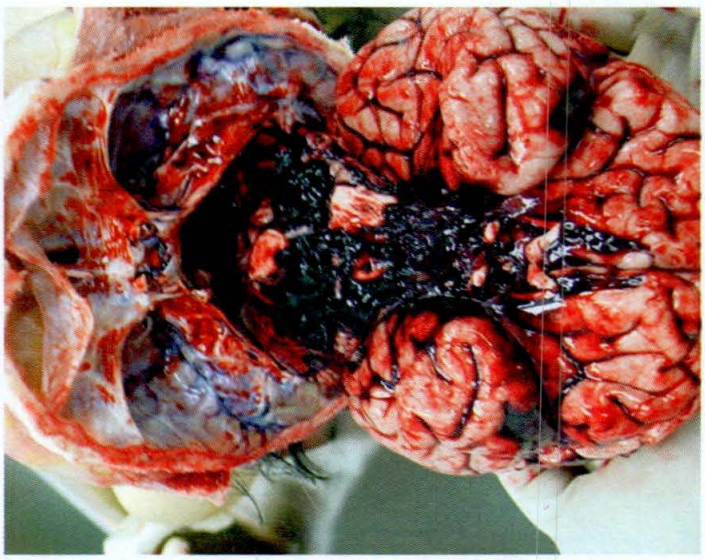


Fig. 78. Subarachnoid haemorrhage caused by rupture of berry aneurysms.

Fig. 79. Subarachnoid haemorrhage.

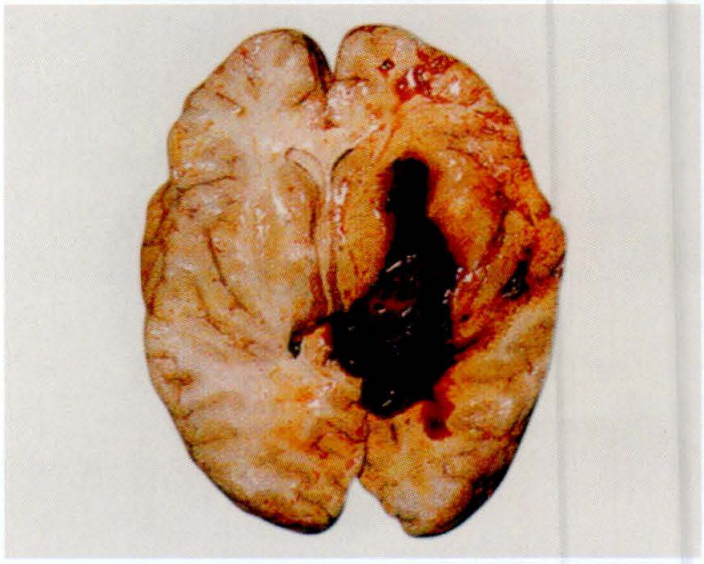


Fig. 80. Haemorrhage in the internal capsule, with extension into the ventricle.

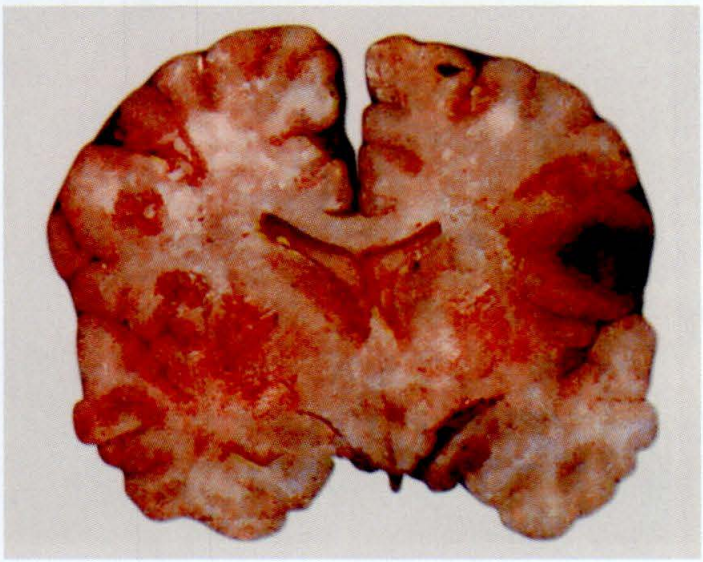


Fig. 81. Intracerebral haemorrhage.

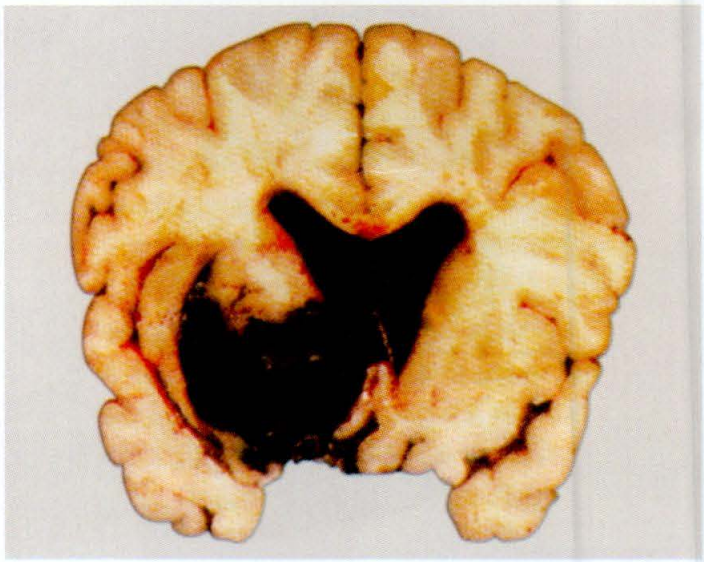


Fig. 82. Haemorrhage in basal ganglia on the left side extending into both lateral ventricles.

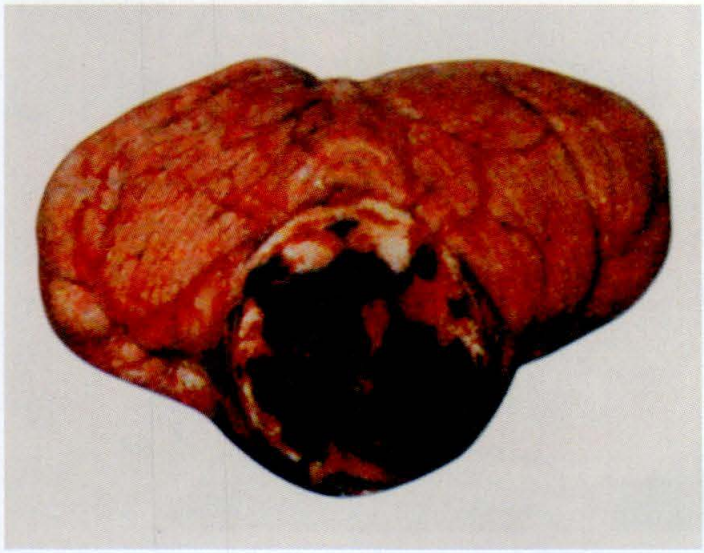


Fig. 83. Massive pontine haemorrhage.

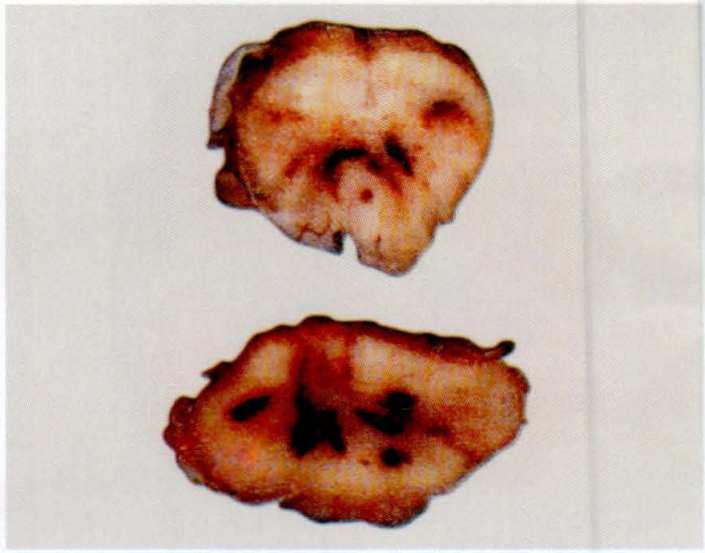


Fig. 84. Haematomyelia.

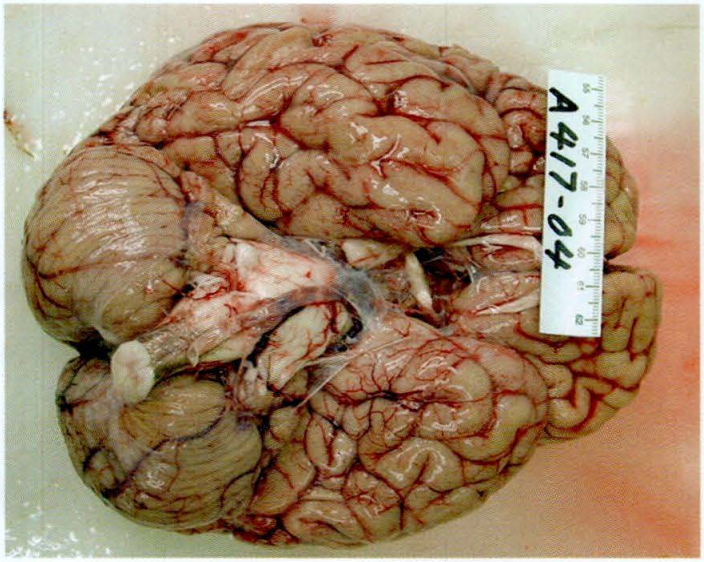


Fig. 85. Oedema of brain showing mushrooming of medulla ablongata and herniation of tonsils.

Fig. 86. Dermo-epidermal burns.

Fig. 87. Various degrees of burns.

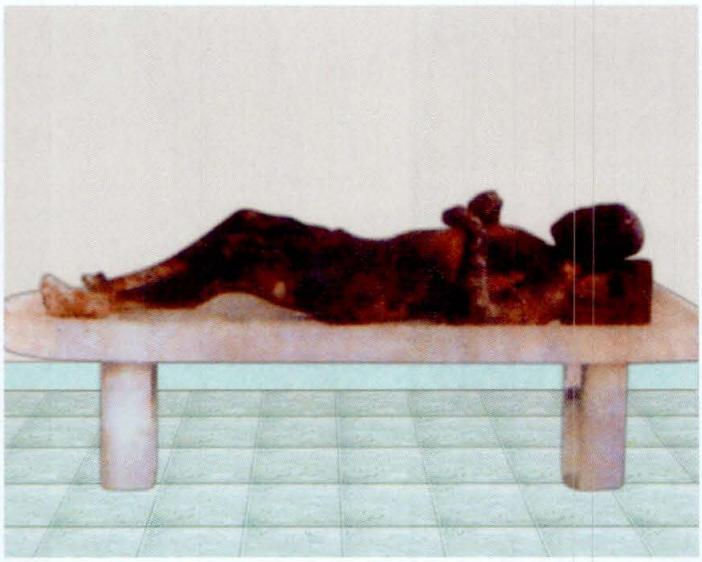


Fig. 88. Puglistic attitude in a case of burns.

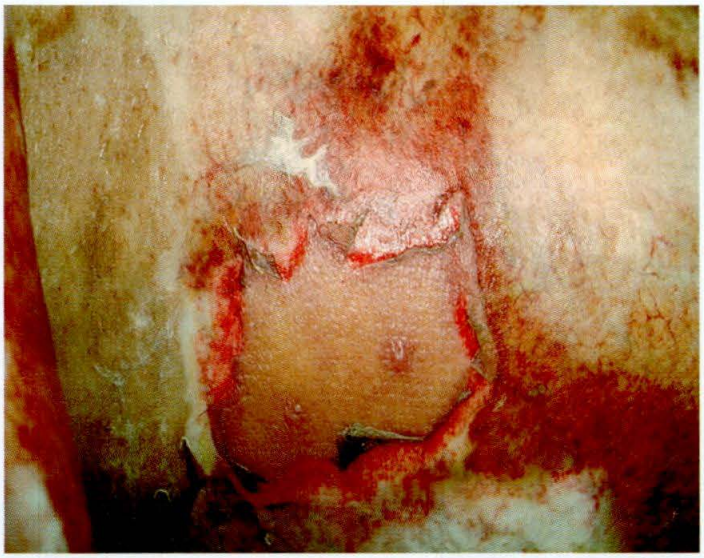


Fig. 89. Antemortem burns showing a zone of hyperaemia at the edge of the burnt area.

Fig. 90. Postmortem burns.

Fig. 91. Extensive burns showing unhealthy granulation tissue after 2 months of survival.

Fig. 92. Heat haematoma.

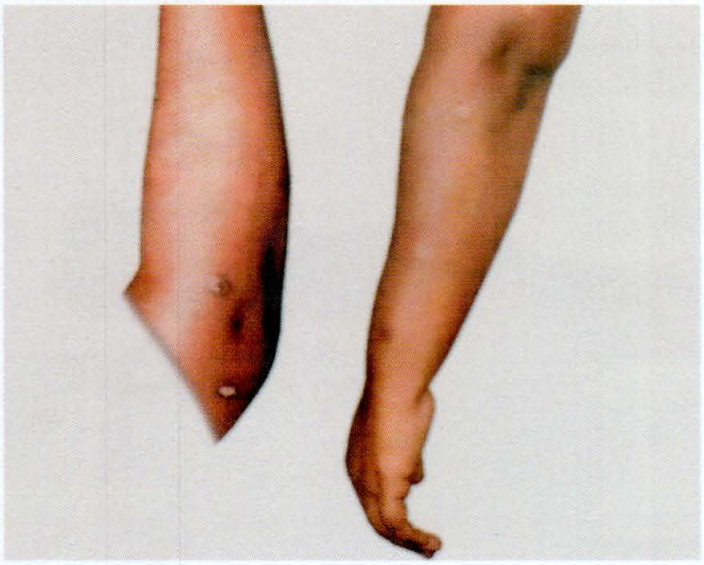


Fig. 93. Cigarette burns on the upper extremity.

Fig. 94. Multiple cigarette burns.

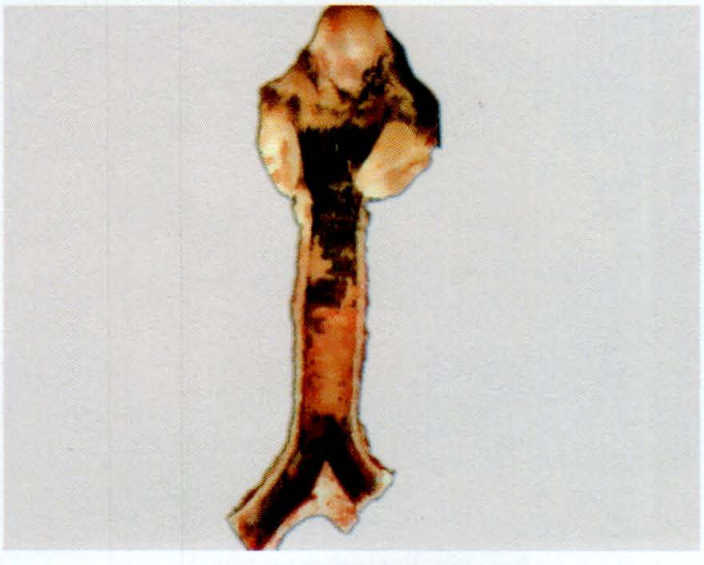


Fig. 95. Soot in the larynx and trachea in case of burns.

Fig. 96. Charred body recovered from a burnt house.

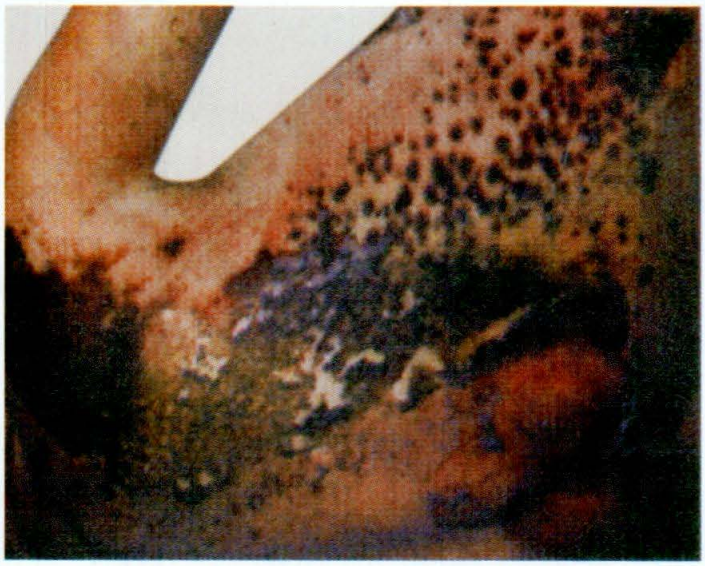


Fig. 97. Crocodile skin appearance in high voltage electric shock.

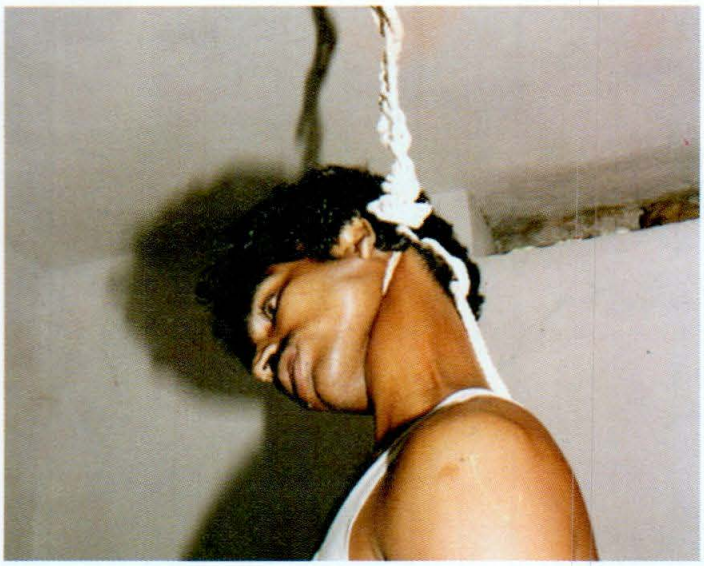


Fig. 98. Suicidal hanging.

Fig. 99. Suicidal hanging.

Fig. 100. Ligature mark of hanging.

Fig. 101. Ligature mark of hanging.

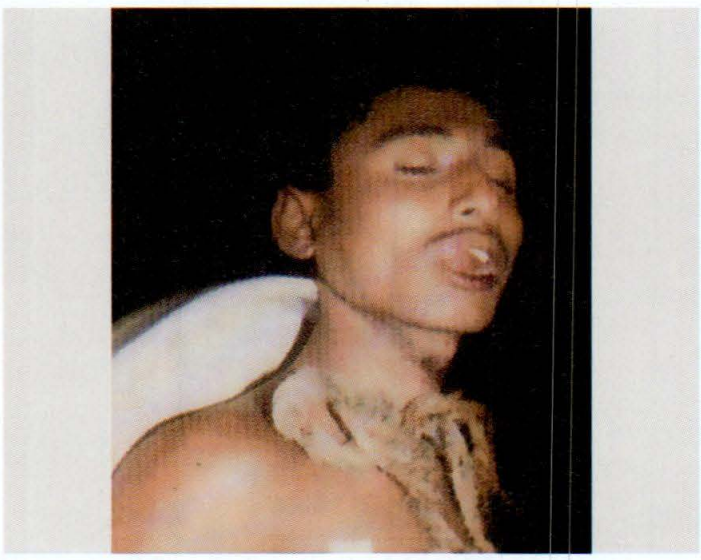


Fig. 102. Ligature mark of hanging corresponding to the ligature used.

Fig. 103. Strangulation by ligature.

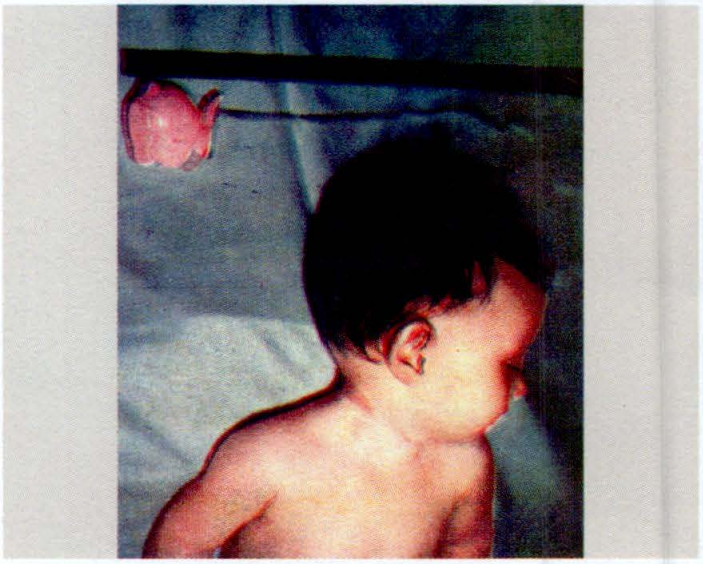


Fig. 104. Homicidal ligature strangulation. Ligature mark produced by the rubber tube of the toy (shown above head) looped around the neck.

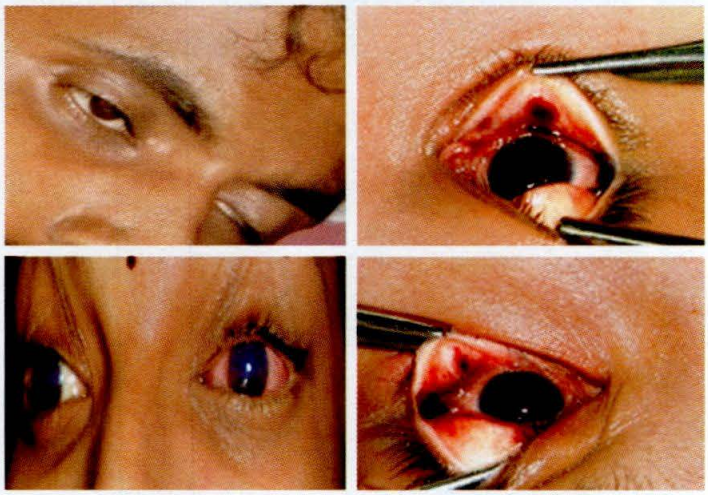


Fig. 105. Eyes in hanging and strangulation.

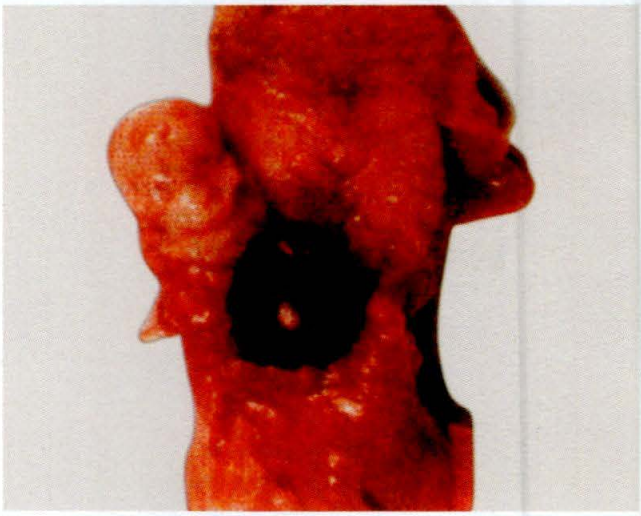


Fig. 106. Chocking from a grape.

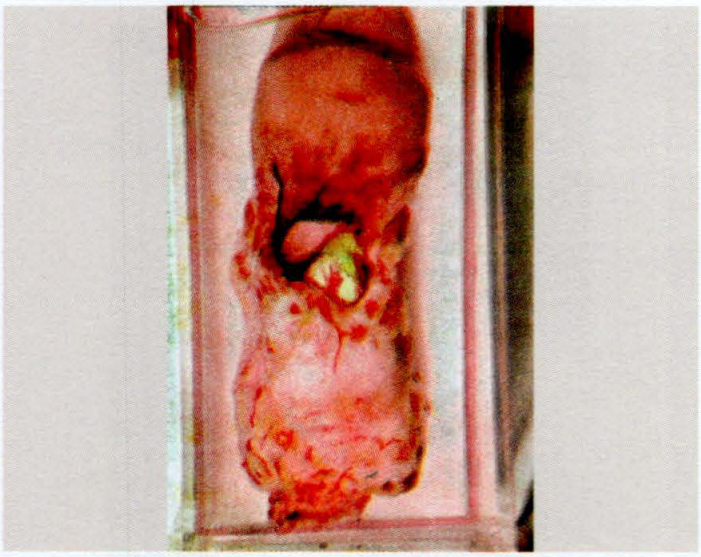


Fig. 107. Chocking due to a bolus of food.

Fig. 108. Traumatic asphyxia caused by fall of a heavy object on the chest.

Fig. 109. Washerwoman hand in drowning.

Fig. 110. Washerwoman feet in drowning.

Fig. 111. Ant bites.

Fig. 112. Rodent bites.

Fig. 113. Spanish fly (Cantharis vesicatoria).

Fig. 114. Heavy metal poisoning.

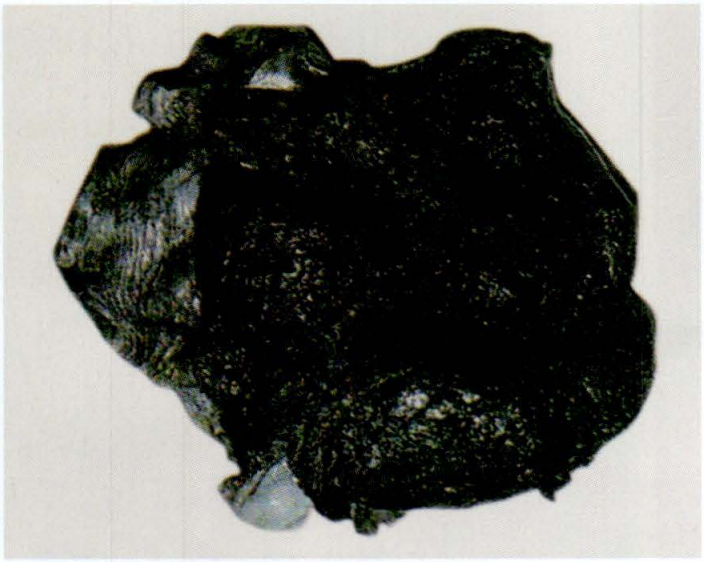


Fig. 115. **Gas gagrene in uterus following criminal abortion.**

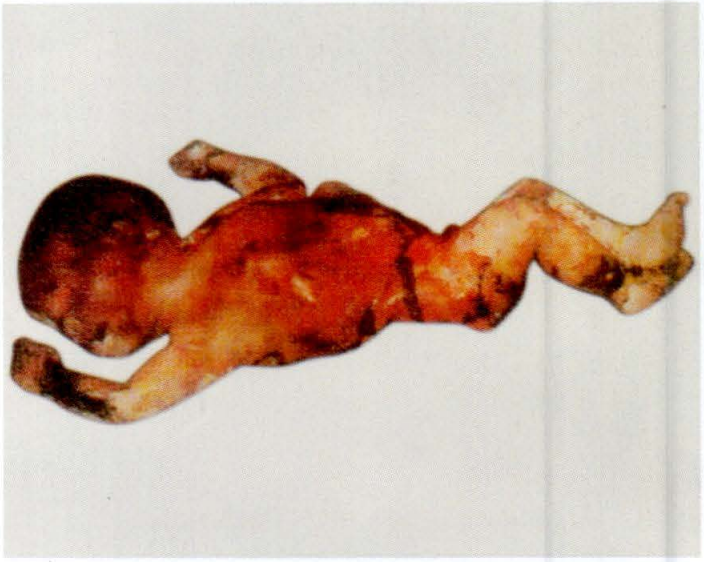


Fig. 116. **Severely macerated foetus.**

Fig. 117. **Haemin crystals.**

Fig. 118. **Haemochromogen crystals.**

Fig. 119. **Vitriolage.**

Fig. 120. **Scorpion.**

CHAPTER

20 Artefacts

Artefact is any change caused, or feature introduced in a body after death, that is likely to lead to misinterpretation of medicolegally significant antemortem findings. Misinterpretation may lead to wrong cause and manner of death and miscarriage of justice. The physician should be aware of the postmortem artefacts, and should be able to distinguish them from the significant antemortem changes.

(A) ARTEFACTS INTRODUCED BETWEEN DEATH AND AUTOPSY: (1) Agonal Artefacts: (A) Regurgitation and aspiration of gastric contents is a common agonal artefact. It may be seen in natural deaths, as a terminal event, or due to handling of the body, or due to resuscitation. (B) Oesophagogastromalacia occurs due to autodigestion, seen rarely in persons dying of head injury with cerebral damage.

(2) Resuscitation Artefacts: (A) The injection marks of resuscitation are usually found in the cardiac region or on the extremities. (B) Some of the injection marks may be associated with postmortem bruises. (C) In intracardiac injection, heart may show contusion and blood may collect in the pericardium. (D) A defibrillator applied to the chest may produce a ring-like contusion. (E) External massage may cause fracture of several ribs and sometimes of sternum. This may be associated with tears of the lungs with release of free air into the tissues. Lacerations of the liver and spleen may occur. In most of these cases, fracture sites do not show bleeding and intercostal muscles do not show contusions.

(3) Artefacts Due to the Handling of the Body: (A) Fresh abrasions may be produced due to dragging of the body during its transfer from the scene of crime. (B) Contusion of the occipital region may be caused, if the head is allowed to fall on a hard surface during handling. (C) Occasionally, fractures of the ribs, or the bones of extremities, or of cervical spine may occur by rough handling of bodies. They are commonly produced during attempts to straighten the limbs contracted due to rigor mortis.

(4) Artefacts related to Rigor Mortis: (A) The handling of the body may cause breaking of the rigor, at least partially, which may mislead the doctor in the estimation of the time of death. (B) Rigor affecting the heart may simulate concentric hypertrophy of the heart. (C) Rigor in pylorus causes it to be unduly firm and contracted.

(5) Artefacts related to Postmortem Lividity: (A) The colour of the postmortem stains is usually bluish-purple. Certain poisons may change the colour of the hypostatic area, e.g., cherry-red colour in CO poisoning, bright-red colour in HCN poisoning, brown or chocolate colour in poisoning by nitrates, potassium chlorate and aniline; dark-brown colour in phosphorus poisoning. (B) Hypostasis in the heart can simulate the effects of coronary occlusion, and in the lungs it may suggest pneumonia. (C) The posterior loops of intestine may become deeply congested and appear dark-red and lumen is filled with bloodstained fluid, giving an appearance of infarction or strangulation. (D) In the occipital region of the scalp, blood accumulates in the soft tissues, which may be mistaken for traumatic haemorrhage.

(6) Artefacts due to Burns: (A) Heat ruptures may resemble lacerated or incised wounds. (B) Heat haematoma may simulate extradural haemorrhage. (C) An unburned groove around the neck due to tightness of the clothes, e.g., collar may resemble a strangulation mark. (D) In severely burned bodies, fat droplets may be found in the pulmonary vessels, which should not be mistaken for antemortem pulmonary fat embolism.

(7) Artefacts in Firearm Wounds: (A) Drainage wounds may be mistaken for firearm wounds. (B) In decomposition, skin may be peeled off with loss of hair and gunpowder from the skin around an entrance wound. The margins of an entry wound may become ragged due to disintegration of the tissue at the margins, and it becomes difficult to distinguish entrance wound from exit. (C) Sometimes, unex-

pectedly a bullet may be found in the body, which may be densely encapsulated, due to old injury, whereas the actual cause of death may be something else.

(8) Artefacts due to Animal and Insect Bites: (A) Rodents bite away tissue over localised areas. They produce shallow craters and scalloping of the borders of the area by nibbling and leave long grooves (Plate 19-112). (B) Marks produced by insects (ants or roaches) are dry, brown, with irregular margins, and are usually seen in moist parts of the body, e.g., ears, armpits, groin, scrotum, anus, etc. They resemble antemortem abrasions (Plate 19-111). (C) Leeches, which become attached to the skin around the eyes, and detach when the body is removed from the water, may produce haemorrhagic lesion simulating a black eye. (D) Crabs and other crustaceans, turtles and fish tend to gnaw the soft tissues around the eyes, ears, mouth, genitals, anus and edges of the surface wounds of the body.

(9) Artefacts in Brain: (A) Regional flattening of the cerebral convolutions is a postmortem artefact, which is seen in those parts of the brain which are in contact with the cranium, especially the occipital lobes. (B) Grooving of the unci is seen in cases of increased intracranial pressure. Uncal grooving is also found in normal brains as an artefact.

(10) Artefacts in Liver: (A) The undersurface of the liver in contact with transverse colon shows greenish colouration due to putrefaction. (B) The liver surface is also stained due to bile.

(11) Postmortem Haemorrhage: (A) Before the blood clots, a postmortem injury may damage a blood vessel and produce haemorrhage. (B) Blood may collect in the pleural cavities due to wounds produced on the chest wall and the lung tissue.

(12) Artefacts related to Hair: The beard may appear to grow after death in some cases, whereas the growth of hair stops immediately after death. The cause of this postmortem apparent growth of beard is due to the shrinkage of the skin, due to which greater part of the hair shaft is exposed above the epidermis.

(13) Artefacts due to Decomposition: (A) Due to decomposition, gases collect in the tissues, cavities and hollow viscera under considerable pressure, and subcutaneous tissues become emphysematous. These changes may produce a false impression of antemortem obesity. (B) Blebs formed due to putrefaction may be mistaken for blebs from burns. (C) Fissures or splits in the skin formed due to putrefaction may simulate antemortem lacerations or incised wounds. (D) Bloody fluid may be found in the mouth and nose in decomposed bodies. (E) Regurgitated gastric juices may cause tanning of the skin of the face and neck, which may simulate antemortem burning. (F) A deep groove simulating ligature mark of strangulation may be seen around the neck in decomposed bodies, if the deceased has been wearing buttoned shirt at the time of death. (G) Intense localised staining of skin due to hypostasis, or displacement of internal pools of blood by pressure of gases of decomposition produce pseudobruising. (H) Separation of sutures of the skull in a child due to gases of decomposition within the brain, and bursting of the abdomen with protrusion of the abdominal viscera due to advanced decomposition should not be mistaken for trauma. (I) Small round holes produced by maggots may simulate bullet holes. (J) Accumulation of blood in the tissues of the neck in drowning may simulate antemortem haemorrhage due to strangulation. (K) Internal hypostasis with haemolysis of red cells may resemble haemorrhage, especially in the meninges, kidneys and retroperitoneal tissues. (L) Rarely, the gastric juice present at the time of death continues to act for some time, causing postmortem digestion of the entire thickness of wall. (M) The blood becomes darker in decomposition due to which the brain, lungs, heart, etc. appear congested, which may be mistaken for signs of asphyxia. (N) Air in the right side of the heart due to decomposition may be mistaken for air embolism. (O) Excessive flaccidity of vaginal orifice, with effusion of bloody fluid and spontaneous detachment of portion of mucous membrane of vagina may simulate antemortem sexual assault.

(14) Artefacts due to Chemicals: In automobile accidents or airplane crashes, exposure to gasoline causes postmortem detachment of epidermis. On exposure to air, the underlying dermis turns peculiar yellow to brown as drying occurs. They resemble thermal burns or abrasions.

(15) Artefacts due to Refrigeration: Pink hypostasis is seen in bodies kept in cold storage.

(16) Embalming Artefacts: (A) The trocar wound may simulate a stab wound. Some blood may be forced

out of injured blood vessels due to pressure and collect in the tissues, and may be mistaken for antemortem haemorrhage. (B) A homicidal stab wound may be enlarged by the embalmer to approach an artery or he may pass a trocar through a gunshot wound. This will modify the dimensions of the wounds. (C) The trocar may also disturb the track of the weapon or bullet and produce false tracks.

(17) Interment and Exhumation Artefacts: In bodies which have been buried, fungus growth is usually seen at body orifices, eyes and at the sites of open injuries. After the removal of the fungus, the colour of the underlying skin resembles bruising.

(18) Toxicological Artefacts: (A) Faulty technique in collecting the sample, especially blood sample, can give erratic results. (B) If blood is contaminated with pericardial or pleural fluids, false results are obtained as regards alcohol, because significant diffusion of alcohol occurs after death, from the stomach to the pleural and pericardial fluid. (C) Certain anticoagulants used for blood, e.g., formalin, heparin, methenamine and EDTA give a positive test for methanol. (D) Blood from embalmed bodies also contain methanol. Decomposition of the tissues after death produces ethyl alcohol and other higher alcohols. (E) Decomposition also causes an increase of concentration of CO in the blood up to nineteen percent. (F) Significant amounts of cyanide are also produced due to decomposition. (G) In cases of death due to burns, significant amounts of cyanide may be found in the blood, possibly due to inhalation of HCN. (H) In buried bodies arsenic may be imbibed from the surrounding earth.

(B) ARTEFACTS INTRODUCED DURING AUTOPSY: (1) Air in Blood Vessels: (A) Pulling of the dura in the sagittal line will cause the air to enter the blood vessels at the top of the brain. (B) Similarly, air may enter the veins of the neck during the reflection of the skin. This may lead to wrong diagnosis of air embolism.

(2) Skull Fractures: Fractures of skull usually in the middle fossae may be produced due to partial sawing and forceful pull of the skull cap, or due to partial sawing and then using chisel and hammer to loosen the skull cap. This may produce additional fractures or may cause extension of already present antemortem fracture.

(3) Visceral Damages: (A) Rough handling of the brain during removal may produce tears of the midbrain. (B) Similarly, rough handling of the liver during removal may produce tears of the diaphragmatic surface.

(4) Extravasation of Blood: (A) If blood has not been drained from vessels of the head, damage to the dura and the dural venous sinuses on removal of skull cap may lead to an escape of blood into the subdural space, simulating an antemortem subdural haemorrhage. (B) Large blood vessels may be cut while opening the thoracic and abdominal cavities, and considerable amount of blood escapes into the pleural and peritoneal cavities. Air may be drawn back into the circulation and enter coronary vessels and give false impression of air embolism. (C) During autopsy, the handling of organs and the incision of the vessels may result in extravasation of blood into the tissues. (D) The removal of the neck structures *en masse* as in routine autopsies may produce artefacts in the neck tissues, which resemble bruises seen in the case of throttling.

(5) Fracture of Hyoid Bone: When the tongue and neck structures are firmly grasped and pulled upon while removing the neck organs, the hyoid bone and thyroid cartilage may be fractured, especially in old persons.

(6) Injury to Blood Vessels: While dissecting the neck structures, if toothed forceps is used, it may damage the intima of the carotid artery which resembles a tear, as is seen in case of strangulation.

(7) Toxicological Artefacts: They may be introduced: (A) due to contamination of viscera with stomach contents during autopsy, or by putting all the organs in one container, or by using contaminated instruments or containers. (B) Faulty technique in collecting the sample. (C) Faulty storage or use of preservatives.

CHAPTER

21 Forensic Science Laboratory

Forensic science is the study and application of scientific examination and evaluation of evidences for legal purposes.

Forensic sciences include: (1) Forensic medicine. (A) Forensic pathology. (B) Forensic psychiatry. (2) Forensic toxicology. (3) Forensic immunology. (4) Forensic odontology. (5) Forensic anthropology. (6) Forensic police sciences. (A) Criminalistics, wherein evidence, such as blood stains, glass, soil, clothing, firearms, etc., is identified, compared, individualised and interpreted; (B) Questioned documents examination involves the scientific examination of handwriting, typewriting, printing, ink, paper, or other aspects of a document for the purpose of determining various legal questions asked about the document. (C) Trace evidence. (D) Ballistics. (7) Other forensic science specialities, which include voice-print examination, polygraph technology, fingerprinting, etc.

Functions: (1) To examine, compare and evaluate physical evidence so as to link a suspect to the victim or to the scene of a crime. (2) Protection of the innocent, e.g., a person arrested for selling narcotics is set free, if the chemical analysis of the material shows it to be harmless. (3) Training of the police investigators as to what constitutes physical evidence, how it is to be found, collected, preserved and delivered to the laboratory.

CRIMINAL INVESTIGATION

PHYSICAL OR TRACE EVIDENCE: All criminal investigation is concerned either with people or with objects. **Only people commit crimes, but they invariably do so through the medium of material objects. It is these material objects, which constitute the broad field of physical evidence. The term physical evidence (trace evidence) includes any and all objects, living or inanimate, solid, liquid or gas, and the relationship between all objects as they relate to the problem in question, e.g., a crime.** A knife, burglar tool, firearms, bullets, blood, saliva, pus, milk, seminal stains, signature, poisons, fingerprints, hair, fibres, glass, paint, oil, dust, signature, microscopic fragments of all types, bacteria, etc. and even an odour are all physical evidence. Physical evidence is useful in two ways: (1) It is often the decisive factor in determining guilt or innocence. It can do this by supplying the demonstrable facts, thus removing differences in ordinary testimony. (2) It can be a material aid in locating the criminal. Evidence should be marked or labelled so that it can be positively identified.

Locard's Exchange Principle: When any two objects come into contact, there is always a transfer of material from each object on the other. Traces from the scene may be carried away on the person, clothes or tools of the criminal, and at the same time, traces from all or any of these may be left at the scene. Wherever a criminal goes, whatever he touches, and whatever he leaves will serve as silent evidence against him, e.g., fingerprints or footprints, hair, fibres from clothes, broken glass, tool marks, paints, scratches, blood or seminal stains, etc. **It is the actual evidence and its presence is absolute proof of the crime. The evidence of eyewitnesses may be wrong as a result of their bias, faulty memory or defective observation. Physical evidence cannot be wrong and completely absent.**

The following are some of the illustrations of the usefulness of forensic science laboratory in criminal investigations. The following paragraphs should be correlated with the appropriate chapters.

ILLUSTRATIONS: (1) Personal Identity: Fingerprints, footprints, hair, blood, semen, etc., are unique to the individual. The criminal may be identified indirectly through the tool he used, the gun he fired, the clothes he wore, writing he made, the soil, glass, paint, etc., he removed from the scene of crime.

(2) Blood: In murder, assault, rape, etc., blood from the victim may be present at the scene of crime and on the accused's person, clothing and weapons. The distribution and appearance of bloodstained areas on the victim and his clothing may be used to interpret and reconstruct details of the crime. The criminal may be injured in the course of struggle or accidentally either

by fall while moving in the dark or by protruding nail or window glass in the act of breaking into a house. Blood groups are very useful in cases of disputed paternity.

(3) Semen: Stains may be found on the accused and victim's clothes, pubic hair and person, on the bedding, mattress, floor or ground on which the offence was committed.

(4) Firearms: A bullet recovered from a dead body can be examined to determine the type of gun which fired it, and the type of ammunition fired. By careful study of the markings on the bullet, the gun which fired it can be determined.

(5) Fingerprints: A criminal can be identified especially by means of latent prints left at the scene of a crime, on a weapon, etc. Grenz rays are useful in many fingerprint investigations, especially when used in conjunction with polaroid papers.

(6) Hair: Hair from a criminal may be pulled out by his victim or attach themselves to the victim or objects at the scene. Similarly, hair from the victim may adhere to the criminal's person, clothing or weapon. In a sexual offence, hair from the victim may be found on the culprit's genitals and vice versa. Hair found sticking to a motor vehicle involved in an accident is useful in the identity of vehicle. Animal hair are very important in case of bestiality, and cattle thefts.

(7) Fibres: A crime against person often involves contact between the criminal's clothes or weapon on one side, and the victim's clothes on the other. Even in burglary, or theft, the criminal often handles or touches several objects in the premises, due to which clothing fibres are transferred from the criminal to the scene of crime and vice versa, because the clothes constantly carry loose fibres. If it is found that a fibre from one source exactly matches one from another source, there is definite probability that the two sources have come into contact with each other.

(8) Poisons: In a suspected case of poisoning the identification of the poison is necessary.

(9) Clothes: Fibres, paint, grease or dust may be found on the suspect's clothes in a burglary, and stains of semen or blood on the clothes of both the victim and the assailant in sexual offences.

(10) Weapons and Tools: A wide variety of tools are used in the commission of crimes, e.g., knives, screwdrivers, bars, saws, pliers, cutters, hammers, drill, etc. Some of these leave marks which are very characteristic, and by which the tool may be quite accurately identified. The tool may also carry traces from the scene in the form of paint, oil, particles of wood or scratches from nails or other hard objects, and these may lead to the detection of crime. The examination of the wound will indicate the type of the weapon used.

(11) Glass: In hit-and-run traffic accident, cyclist's back lamp may be broken, the glass fragments of which may be found on bumper or other parts of the motor vehicle. Also, traces of glass from the headlight of the vehicle may be found at the scene. The burglar in trying to enter the house may break window glass, the fragments of which may be carried in his clothes, etc.

(12) Wood: If a piece of wood from the handle of the tool used by the criminal is found at a scene, it can be identified by matching it with the handle of the tool recovered from the suspect. Particles or splinters of the wood found on the suspect's clothes or tool should be compared with the wood of the door or window broken for the forced entry.

(13) Metals: Metal pieces from the tool used by the criminal may be found at the scene of the crime, and metal fragments from the door and window fittings and from the boxes and safes, may be recovered from the criminal's clothing or tool.

(14) Paint: In a road accident, flakes of paint from the vehicle may be found on the ground or on the person, animal or object hit by it, and traces of paint from the object may be seen on the vehicle. A burglar may carry on his body, tool or clothing, paint from the wall or doors of the house which he has burgled or from the safe which he has broke open.

(15) Dust and Dirt: A criminal invariably carries soil in varying quantities on his feet or footwear from the earth on which he has walked, and on his body, or clothing from the ground on which he lied down during the commission of the crime, or fallen in the course of a struggle. Soil usually consists of mineral constituents, decomposed organic matter, broken leaves, pollen, grains, etc., capable of direct identification.

(16) Vegetable Material: Anyone committing a crime out of doors is likely to get plant material on his clothing, the identification of which will connect him with the scene. Algae and fungi are usually found on damp walls, in buildings, the soils, on vegetation and on domestic articles. The burglar climbing the damp wall or fall-pipe may get smears of green algae. Seeds, portions of leaf, bark and other vegetable fragments are useful and can be identified to belong to particular areas of country. Grass fragments, particularly uncommon types, pollen, weeds and seeds are identifiable with their particular source of origin.

(17) Strings and Ropes: The criminal may have brought his tools to the scene tied up in a bundle with string or cord and may have left it behind. If it is identical in structure, size, shape and appearance with another found in the suspect's possession, it is of great value.

(18) Tyre Marks: It may be possible to trace a car by means of its tyre marks.

(19) Documents: Questioned documents may necessitate: (1) Physical and chemical examination including a study and identification of: (a) writing materials, e.g., paper, pen, ink, pencil, typewriter, (b) erasures, obliterations and alterations, (c) order and age of writing, typing or other markings. (2) Identification of the authorship of the writing. Indented writing is made out by using very oblique lighting and photographing it. Transmission electronography shows structure of paper and watermark, and the Grenz-ray technique shows all the surface detail and watermark.

(20) Photography: In the forensic sciences photographs are used (1) as a means to record what has been observed, and (2) to reveal that cannot normally be seen.

(21) Lie Detection: (A) Polygraph: It is an instrument used to detect lies. Polygraph makes a continuous record of blood pressure, pulse, respiration and electrodermal reaction changes in response to stimuli in the form of questions. It is based on the theory that when the person tells a lie in answer to a question and there is fear that lie will be detected, the fear results in stimulation of sympathetic nervous system which results in certain physiological changes (psychosomatic reaction), some of which may be easily recorded. There is relative rise in blood pressure and recovery, slowing down of the breathing and many times suppression and lowering of the skin resistance of the individual due to increased resistance of the sweat glands. In pre-test interview, the test questions are framed with the mutual consent of the person to be examined and to the satisfaction of examiner, that they are adequate to serve the purpose of the particular examination. A basic explanation of the attachments in the polygraph is given to the person. An attempt is made to answer the person's questions regarding the procedure. The questions are framed in such a way that they are clearly understood by the person, and they call for only "Yes" or "No" as answer. **The questions usually number ten. Relevant and irrelevant questions are mixed up.** The control questions are put to reduce the natural nervousness, the natural disgrace of the issue at stake, and the natural slight resentment of the accusatory nature of the matter involved in the investigation. **A question is asked every 20 to 25 seconds, and polygraph chart recorded in three to four minutes.** Usually the same test is repeated twice or thrice as a check on any possible error. An experienced and competent polygraph examiner can correctly detect truth or lie in about 80 to 90% cases. Offenders, suspects, complainants, witnesses and informants are examined by this method to test truth of their statements. It is also useful in civil cases, e.g., paternity cases, insurance claims, pre-employment screening by banks and other institutions.

BRAIN MAPPING (BRAIN FINGER PRINTING): It is a technique that measures recognition of familiar stimuli by measuring electrical brain wave response (P300) to words, phrases or pictures that are presented on a computer screen. It is based on the theory that the suspect's reaction to the details of an event or activity will reflect, if the suspect had prior knowledge of the event or activity.

Technique: The equipment called **"electrocap"** is fixed on the suspect's head for recording EEG. The suspected person is questioned about the crime and also shown the visuals of the crime scene, such as victim, weapon, time, place and how he committed the crime relevant to a crime, along with irrelevant words, photographs, etc., to stimulate his brain and encourage a reaction, on a computer monitor. Apart from his verbal replies, another computer keeps track of the neuro impulses (brain wave) chemical responses emitted whenever the visual is seen. **The intensity of the brain-waves shoots up whenever a question or the visual stimuli matches the information stored in the brain, if the suspect is really the perpetrator of the crime.** This can be tracked and printed to provide the best scientific evidence. **It depends only in information stored in the brain and cognitive brain responses and does not depend on the emotions and is not affected by emotional responses.** Similarly, when the information tested is known only to members of a particular organisation/group, e.g., terrorist group, the information present indicates affiliation with the group in question. **It is said to be 100% accurate.**

(B) Narcoanalysis ("Truth serum" drugs): The underlying principle is that at a point very close to unconsciousness, the person will be mentally incapable of resistance to questioning and incapable of inventing the falsehoods that he has used to conceal his guilt. **The drugs will remove inhibitions but not**

self-control. The methods used are: (1) Half milligram of scopolamine hydrobromide s.c., followed by one-fourth mg. every 20 minutes for an average of 3 to 6 injections, until the person reaches the proper stage for questioning. (2) Sodium amytal or sodium pentothal (truth serum) 2.5 to 5% solution i.v., at a rate not to exceed 1 ml/m., until the proper stage is reached. **It depresses CNS and person loses inhibitions. In a state of relaxation, the suspect is susceptible to suggestion and reveals repressed feelings or memories.**

(C) Hypnosis: Many people can be hypnotised and many cannot be hypnotised to a deep level. It does not often enhance memory. Hypnotised witnesses (1) Produce more fabricated recollection, (2) are more influenced by interviewer's misleading comments and questions, and (3) more confident in the accuracy of their recollections, than are nonhypnotised witnesses, even when their recollections are false.

Consent: In India, no person should be forcibly subjected to narcoanalysis, polygraph and brain-mapping tests on accused, suspects and witnesses without their consent as they violate the right against self-incrimination. Even when the person gives consent, they will not be admitted as evidence in a court.

CHAPTER

22 Forensic Psychiatry

Psychiatry deals with the study, diagnosis, and treatment of mental illness. **Forensic psychiatry** **deals with application of psychiatry in the administration of justice. Mental illness or unsoundness of mind can be defined as a disease of the mind or the personality, in which there is derangement of the mental or emotional processes and impairment of behaviour control.** The intelligence is weakened or perverted, but the insane person may not show physical weakness. The law has not defined insanity. **The term is used for those persons who are unable to adopt themselves to the ordinary social requirements due to mental disease. The law is most frequently concerned with "mental impairment" and not "mental illness".** Different kinds and degrees of mental impairment are required for different legal issues.

Common terms used in psychiatry:

AFFECT: Emotion, feeling or mood.

AFFECTIVE DISORDER: Psychiatric disorder in which the chief feature is a relatively prolonged affective change of an abnormal degree, i.e., depression, mania.

APHASIA: The loss of ability to express meaning by the use of speech or writing (motor aphasia), or to understand spoken or written language (sensory or auditory aphasia).

DELIRIUM: It is a disturbance of consciousness in which orientation is impaired, the critical faculty is blunted or lost, and thought content is irrelevant or inconsistent. In the early stage, the patient is restless, uneasy and sleepless. He then completely loses self-control, becomes excited and talks furiously. Delusions and sometimes hallucinations are present. It usually occurs in physical diseases in which there is continuous high temperature and sometimes due to overwork, mental stress, metabolic diseases, cerebral neoplasms or drug intoxication. A person may become impulsive and violent and may commit suicide. **Such person is not responsible for his criminal acts.**

DELUSION: Delusion is a false belief in something which is not a fact, and which persists even after its falsity has been clearly demonstrated. A normal person can have a delusion, but is capable of correcting it by his reasoning power, by his past experience and by being convinced by others. Delusion in insane person is a symptom of brain disease. **It is under the control of emotional but not rational forces.**

Types: (1) Grandeur or exaltation: A man imagines himself to be very rich while in reality he is a pauper. **(2) Persecution (paranoid):** The person imagines that attempts are being made to poison him by his nearest relatives like wife, sons or parents. Delusions of grandeur and persecution are often present together in the same person. **(3) Reference:** The person believes that people, things, events, etc., refer to him in a special way. He believes that even strangers in the street are looking at him and are talking about him or items in the radio or newspapers are referring to him. **(4) Influence:** The person complains that his thoughts, feelings and actions are being influenced and controlled by some outside agency, like radio, hypnotism, telepathy, etc. **(5) Infidelity:** A man imagines his wife to be unfaithful while in fact she is chaste. **OTHELLO SYNDROME:** The person has delusion of infidelity about his wife or mistress and he assaults her. If she confesses due to severe pressure, he explodes into violent acts and he may attempt or commit murder. **(6) Self-reproach:** The person blames himself for the past failures and misdeeds which are often of no importance. **(7) Nihilistic:** The person declares that he does not exist or that there is no world, etc. **(8) Hypochondriacal:** The person believes that there is something wrong with his body, though he is healthy. **(9) Other types** are of jealousy, of religion, etc.

M.L. Importance: Delusion is never an isolated disorder, but is an indication of deep-seated, widespread brain disorder. For this reason, **such person cannot be regarded as fully responsible for his antisocial acts.**

EROTOMANIA: Erotomania is a delusion in which the person believes that someone is deeply in love with him/her. The erotomanic develops an obsession for a particular person and starts believing that the other person is reciprocating. The person is usually of a higher status, famous or superior at work, but can also be a complete stranger. The erotomanic tries to get close with the person through telephone calls, letters, gifts, visits, etc. The person is otherwise normal.

FUGUE: A state of altered awareness during which an individual forgets part or whole of his life, leaves home and wanders.

HALLUCINATION: Hallucination is a false sense perception, without any external object or stimulus to produce it. They are purely imaginary and may affect any or all the special senses.

Types: (1) Visual: A person imagines of being attacked by a lion when no lion exists. **(2) Auditory:** A person hears voices and imagines that a person is speaking to him when no one is present. **(3) Olfactory:** A person smells pleasant or unpleasant odour when none is present. **(4) Gustatory:** A person feels sweet, sour, bitter, good or bad taste in the mouth, though no food is actually present. **(5) Tactile (Haptic):** A man imagines rats and mice crawling into his bed, when there are none. **(6) Psychomotor:** A man will have feeling of movement of some part of the body in the absence of such movement. **(7) Command:** The patient is ordered by hallucinatory voice to do things/acts, which may be frightening or dangerous. (8) **Microptic or macroptic:** Any object appears usually smaller or bigger. (9) **Sexual:** A person feels sexually satisfied from unfounded self-imaginative objects.

Hallucinations occur in fevers and intoxications, as well as insanity. Visual and auditory hallucinations are the most common. They may be pleasant, but more often they are unpleasant. A person suffering from unpleasant hallucinations may commit suicide or homicide.

ILLUSION: Illusion is a false interpretation by the senses of an external object or stimulus which has a real existence, e.g., (1) when a person sees a dog and mistakes it for lion, (2) hears the notes of birds and imagines them to be human voices, (3) imagines a string hanging in his room to be snake, (4) may mistake the stem of a tree for a ghost in the dark. A sane person may experience illusions, but is capable of correcting the false impressions. An insane person continues to believe in the illusions even though the real facts are clearly pointed out.

IMPULSE: This is a sudden and irresistible force compelling a person to the conscious performance of some action without motive or forethought. A sane person is capable of controlling an impulse. An insane person having no judgment and no reasoning power and no capacity to understand the facts, may do things on impulse. These are usually seen in imbecility, dementia, acute mania, and epilepsy.

Types: (1) Kleptomania: An irresistible desire to steal articles of little value. **(2) Pyromania:** An irresistible desire to set fire to things. **(3) Mutilomania:** An irresistible desire to mutilate animals. **(4) Dipsomania:** An irresistible desire for alcoholic drinks at periodic intervals. **(5) Sexual impulses:** Compulsive urge to perform sexual intercourse which may often be in a perverted way. **(6) Suicidal and homicidal impulses.**

OBSESSION: In this, **a single idea, thought or emotion is constantly entertained by a person which he recognises as irrational, but persists in spite of all efforts to drive it from his mind.** It is a disorder of content of thought. Any attempt to resist makes them appear more insistent. It is a borderline between sanity and insanity. They usually occur in neurotic people, well able to discharge the ordinary responsibilities of life. These ideas are usually associated with some sort of dread or fear. A wife may continuously believe her husband to be unfaithful in spite of proof to the contrary. A person may go to bed at night after securely bolting the door of his room, but he soon gets up to see he has done so. A sane person may repeat it once or twice, but an insane person does not sleep, and spends the whole night in frequently seeing whether the door is bolted or not.

PHOBIA: It is an **excessive or irrational fear of a particular object or situation.**

LUCID INTERVAL: This is a period occurring in insanity during which all the symptoms of insanity disappear completely. The individual is able to judge his acts soundly, and he becomes legally liable for his acts. If he commits an offence, he cannot be completely held responsible, because it is

Table (22-1). Difference between lucid inerval in insanity and head injury

Trait	Insanity	Head Injury
(1) History:	Of insanity present.	Of injury to the head.
(2) Preceding symptoms:	Of insanity.	Of concussion.
(3) Following symptoms:	Of insanity.	Of cerebral irritation and compression of the brain.
(4) Occurrence:	Frequent.	Only once.

very difficult to know whether he was suffering from some mental abnormality at the time of committing the offence. In mania and melancholia, lucid intervals are common (Table 22-1).

ONEIROID STATES: (oneirophrenia): **It is a dream-like state which may last for days or weeks.** The patient suffers from mental confusion, amnesia, illusions, hallucinations, disorientation, agitation and anxiety. It occurs in delirium and early schizophrenia.

TWILIGHT STATE (Psychomotor automatism): **It is a state of diminished awareness of acts of relatively short duration of which he has no recollection. The patient may do some unaccustomed automatic acts, sometimes in an aggressive way and may suffer from visual hallucinations. It is usually seen in epilepsy.**

Hysteria: It is characterized by self-dramatisation, excessive emotionality and attention seeking behaviour. Egocentric and demanding interpersonal relationships are typical of this condition.

PSYCHOPATH: **Psychopath is a person who is neither insane nor mentally defective, but fails to conform to normal standards of behaviour.** Psychopaths have abnormal personality, persistently behave in an antisocial or disruptive manner, and are unable to appreciate the normal implications of their actions.

In this there is a failure of maturation of the personality, the individual retaining a child-like selfishness. They have no abnormality of thought, mood or intelligence, but their behaviour is unacceptable. Lack of emotional response, constant desire for the gratification of their desires, and complete lack of conscience are characteristic of psychopathic personality. Immediate violence including murder can arise from any challenge to their egotism. They can plan and implement their antisocial acts in an efficient way. **The basic defect appears to be moral, rather than psychological or neurological.**

PSYCHOSES: **They are characterized by a withdrawal from reality; a living in a world of fantasy** (Table 22-2).

Table (22-2). Difference between psychosis and neurosis

Trait	Psychosis	Neurosis
(1) Nature:	A disease entity with a physical basis which is determined genetically.	A reaction to stressful circumstances due to adverse childhood experiences.
(2) Severity:	Major.	Minor.
(3) Empathy:	Absent.	Present.
(4) Contact with reality:	Absent.	Present.
(5) Insight:	Absent.	Present.

NEUROSES: **The patient suffers from emotional or intellectual disorders, but he does not lose touch with reality.** They occur mostly in the form of anxiety, depression or hysteria (Table 22-2).

NEURASTHENIA: **It is a condition of nervous exhaustion due to physical or mental conditions. There is abnormal fatigue and irritability of the nervous system.**

MUTISM: **It is complete loss of speech. It is seen in hysteria, catatonic schizophrenia, depression, organic brain lesion and malingering.**

CAUSES OF INSANITY: **(1) Hereditary, e.g. Huntington's chorea and amaurotic family idiocy. (2) Environmental factors, e.g., faulty parental attitude and lack of mental hygiene. (3) Psychogenic, e.g., unsuccessfully repressed mental conflicts. (4) Precipitating, e.g., financial and business worries, frustrations and disappointments in sexual affairs, death of close relatives, senile degeneration, myxoedema, pernicious anaemia, etc.**

CLASSIFICATION

The World Health Organisation has recommended in the International Classification of Diseases (1992) the following:

(a) Organic mental disorders include dementia in Alzheimer's disease, vascular dementia and dementia

in other diseases, organic amnesic syndrome, delirium not induced by alcohol and other mental, personality and behavioural disorders due to brain disease, damage or dysfunction. (b) Mental and behavioural disorders due to psychoactive substance use. (c) Schizophrenia. (d) Mood disorders. (e) Neurotic stress-related and somatoform disorders. (1) Anxiety disorders. (2) Obsessive-compulsive disorder. (3) Reaction to stress and adjustment disorders. (4) Dissociative (conversion) disorder. (5) Somatoform disorders. (f) Behavioural syndromes associated with physiological disturbances and physical factors. (1) Eating disorders. (2) Non-organic sleep disorders. (3) Sexual dysfunction not caused by organic disorder or disease. (4) Mental and behavioural disorders associated with puerperium. (5) Abuse of non-dependence producing substances. (g) Personality disorders. (1) Specific personality disorders. (2) Habit and impulse disorders. (3) Gender identity disorders. (4) Disorders of sexual perversion. (h) Mental retardation. (i) Disorders of psychological development.

Intelligent Quotient (I.Q.): It is the intellectual capacity of a person in relation to his chronological age, which is expressed as a percentage. Normal adult I.Q. is 90 to 110.

MENTAL RETARDATION: Earlier under the term amentia, three groups: idiocy, imbecility and feeble mindedness (morons) were recognised, which is now replaced by the term mental retardation and includes mental sub-normality and mental handicap. It refers to below average general intellectual functioning with typical onset in infancy or birth that must occur before 18 years of age. It signifies a condition of retarded, incomplete maturation of attention, perception, cognition and social adaptability. It results from environment, genetics, endocrinal, metabolic, infective, toxic causes and birth trauma.

It is graded according to IQ levels as:

1. Mild: IQ 50 to 70
2. Moderate: IQ 35 to 55
3. Severe: IQ 20 to 40
4. Profound: IQ below 25.

PSYCHOSIS ASSOCIATED WITH ORGANIC DISEASES: Mental illnesses which arise from some structural brain damage are called organic psychoses. The organic psychoses are characterised by lability of mood, failure of memory, deterioration of intellect, irritability, irrational anger, confusion, loss of social inhibitions, etc. which may lead such persons into unacceptable behaviour and even sudden violence.

Dementia: Dementia is a condition in which there is degeneration of mental faculties after they have been fully developed. (a) Alzheimer's disease. (b) Pick's disease. (c) Creutzfeldt-Jacob disease. (d) Huntington's chorea.

(1) Presenile dementia.

(2) Senile Dementia: It is caused due to arteriosclerosis and old age. It usually starts after 65 years. A feeling of loneliness, of being unwanted, loss of prestige and death of a close relative cause the onset of the disease. The patient is confused, judgement and memory is impaired. Delusions, hallucinations and emotional outbursts are common.

(3) Cerebral Tumours: Mental symptoms may occur at any stage of growth of cerebral tumours involving prefrontal, frontal, temporal and parietal lobes.

(4) Cerebral Trauma can precipitate any type of mental illness.

(5) Drug-induced Psychosis: Dependence on barbiturates, amphetamines, cannabis, heroin, cocaine, etc. leads to psychosis. Cocaine, LSD, amphetamines and mescaline can produce clinical symptoms similar to schizophrenia.

(6) Toxic Psychosis: Heavy metals, such as arsenic and mercury may produce mental degeneration.

(7) Deficiency States: Deficiency of cyanocobalamin (pernicious anaemia), nicotinic acid (pellagra), 5-hydroxytryptamine (phenyl ketonuria), thyroxine (myxoedema), pituitary hormones (hypopituitarism) and hypoglycaemia produces mental degeneration.

(8) General Paralysis of the Insane: This is a chronic progressive condition leading to paralysis and dementia. It is usually associated with meningovascular syphilis and tabes dorsalis.

(9) EPILEPTIC PSYCHOSIS: Short transitory fits of uncontrollable mania occur. There may be progressive dementia.

(1) Pre-epileptic Insanity: Instead of epileptic aura, the patient may occasionally develop violent fits of mania, or extreme depression of mind. Hallucinations and delusions are common during this stage, and such persons may commit assault or other criminal acts.

(2) Post-epileptic Insanity: The stupor following epileptic fit, is replaced by automatic acts of which the patient has no recollection. The patient is confused and terrified by visual and auditory hallucinations and delusions of persecution, and may commit crimes, such as thefts,

burning of property, sexual assaults and murders. **These crimes are involuntary, automatic and unpremeditated.** The patient never attempts to conceal them at the time of committing, but may try to conceal them on regaining consciousness. **Automatic action tends to be of the same type in each attack.** The action is usually habitual, e.g., a man walks into a shop, picks up something and walks out again, afterwards being arrested for theft; or one who exposes himself in a public place and is arrested for indecent conduct; or a person cutting something may inflict incised wounds or kill a child; or a person accustomed to firearms may shoot somebody.

(3) Psychomotor Epilepsy (temporal lobe, masked, or psychic epilepsy): In this, instead of epileptic fit, the patient suffers from temporary and transient seizure of maniacal excitement with loss of consciousness. The patient may commit crimes without any motive.

FUNCTIONAL PSYCHOSES: Mental illnesses which have no neurological basis are called functional psychoses. They are characterised by disorders of thought which have no physical basis. **(a) In schizophrenic psychoses disorders of thought process are dominant. (b) In affective psychoses mood abnormality is dominant.**

SCHIZOPHRENIA: It is a condition of split-personality, in which the patient loses his contact with his environment. It is primarily a disorder of thinking (cognition).

This disorder can be in form, stream, possession or content of thought. It is characterised by: (1) **Disorders of behaviour:** withdrawal from reality, pre-occupation with the self (narcissism), attribution of a feeling of strangeness to outside influence (depersonalisation), and feelings that his mind and body are under control (passivity of feeling). (2) **Disorders of thought:** confused thoughts leading to thought block, devious thinking leading to incoherence of speech often with newly formed words (neologism). (3) **Disorders of affect:** depression, elation, inappropriate moods, lability of mood, anxiety and blunting of emotions. (4) **Delusions:** of grandeur, paranoid, hypochondriac and influence. (5) **Hallucinations:** commonly auditory, sometimes visual and tactile. (6) **Personality deterioration**, affecting his work, family and social relationships.

It is the commonest type of insanity in homicidal crimes, especially where the victim is a stranger. The impulses are not sudden and the crime is usually preceded by much complaining and planning.

(1) Simple schizophrenia: It begins in early adolescence. There is a gradual loss of interest in the outside world from which he withdraws. There is impairment of mental faculties. He becomes emotionally flat and apathetic and has difficulty in forming social relationships. Complete disintegration of the personality occurs later.

(2) Hebephrenia: It begins in adolescents or young adults. Thinking process is disturbed. Wild excitement, illusions, hallucinations, and delusions are present. Often conduct is impulsive and senseless. Ultimately, the whole personality may disintegrate completely.

(3) Catatonia: This is characterised by alternating stages of depression, excitement and stupor; impulsive suicidal or homicidal attacks and auditory hallucinations are common. This phase lasts for few hours to few days followed by a stage of stupor which begins with lack of interest, concentration and general indifference.

(4) Paranoid schizophrenia (paranoia, paraphrenia): Paranoia is the mild form, characterised by delusions of persecution. It is common in males. Paranoid schizophrenia develops insidiously in the fourth decade. It is characterised by suspiciousness, delusions of persecution, and auditory hallucinations. At first delusions are indefinite but later they become fixed on some person. The patient usually retains his memory and orientation. When delusions affect his behaviour, he is often a source of danger to himself and others.

AFFECTIVE TYPE: These diseases are of hereditary origin affecting young adults and form a major group of all psychiatric illnesses. **(1) Manic-depressive psychosis:** They show wide swings of mood from euphoric elation to deepest depression, quite out of proportion or often totally unrelated to external circumstances. The two extremes of these moods are hyperactive, excitant at one end and depressive stupor at the other. **The primary disturbance is of affect. It occurs periodically. Isolated attacks of mania and depression may occur in the same patient and some show attacks of one type only.**

Manic phase: This is a condition of exaltation of the emotions and the intellect. (A) Acute mania: It is characterised by euphoria or irritable mood, excitement, loss of self-control, flight of ideas and great muscular activity. Mood is elated, attention is fleeting and there is high degree of distraction. **(B) Hypomania:** It is the mildest form in which there is an exaggerated sense of self-importance. Offences, such as petty theft, deception, indecent assault and fraud may be committed.

(2) Depressive phase (melancholia): It is an intense feeling of depression and misery without any cause. The sadness of mood is reflected in

posture, movements and facial expression. He retires from his usual social activities, avoids friends. Suicide is well planned and is of great danger to the patient. He may kill relatives, especially dependent young children. Homicidal and suicidal tendencies co-exist. They have feelings of self reproach and guilt, and marked psychomotor disturbances.

Types: (1) Endogenous. (2) Reactive and neurotic. (3) Involutional. (4) Puerperal.

NEUROSES: They may occur singly or in combination.

(1) Anxiety neurosis: It results from autonomic stimuli. The patient has spells of dyspnoea, choking, palpitation, insomnia, faintness, trembling, headache, chest pain, etc. The person is apprehensive and depressed and worries about his health. Intolerance of noises, children and spouse may be seen.

(2) Hysterical neurosis: It is common in young females, but may occur in old ladies whose nervous system starts degenerating. The symptoms commence when faced with unpleasant life situations. The patient shows deafness, blindness, loss of smell, anaesthesia, paraesthesia, paralysis, aphonia, etc.

(3) Phobic neurosis: Phobias are specific fears, which the patient recognises as irrational. They often act as defence mechanisms that protect the patient from unpleasant life situations. The different types are: acrophobia (high places), agoraphobia (open spaces), claustrophobia (closed spaces), necrophobia (dead bodies), nyctophobia (darkness), phonophobia (sound), pathophobia (disease), thanatophobia (death), toxiphobia (being poisoned), etc. It interferes with daily life.

(4) Obsessive-compulsive neurosis: Anxiety, depression and fear are present in varying degrees. Obsessional thoughts may be associated with a whole range of objects. Doubt may exist whether doors are locked or lights switched off.

(5) Depressive neurosis: It resembles manic-depressive psychosis but the degree of depression is less, and mental retardation, delusions and hallucinations are absent. There is risk of suicide.

PERSONALITY DISORDERS: The person is said to suffer from a personality disorder, when the behaviour is inappropriate to the situation. Psychopathic personality refers to a special derangement of the mind. The important features are: (1) Lack of normal conscience. (2) Absence of normal feelings for other people, such as love, affection, sympathy, etc. (3) A tendency to antisocial impulsive acts. (4) Failure to learn from experience and to be prevented from crime by punishment. (5) Freedom from any other form of mental disorder. The symptoms usually appear in early childhood but become marked in adulthood and gradually diminish in later life. Sexual perversions and crimes are common.

Sexual Deviation: It may be a symptom of an underlying psychiatric disorder, such as schizophrenia, senile dementia, personality disorder. The person indulges in sexual perversions and unnatural sex practices.

Drug Dependence: Drug addicts have a wide range of personalities. Most of them are emotionally unstable, immature, impulsive, angry with society, and unable to achieve their goals, or face difficult situations in life. About 10% suffer from psychopathic personality disorder.

Diagnosis of Insanity: The person should be kept under observation in a general hospital, or nursing home, or psychiatric hospital or nursing home, or in any other suitable place, **which should not exceed 10 days, but with the permission of the Magistrate, he may be detained for further periods of 10 days up to a maximum of thirty days.** Violent and criminal persons should be kept in a prison. The person should be watched during different times of the day, when he is alone, in company and while he is working, eating, reading or writing, and when he is unaware of the fact of being observed.

Certification: A certificate should not be issued after single examination. Three examinations on different days and different hours are usually recommended, with the possibility of more, because a person may behave peculiarly at a single examination, either due to the effect of drugs or due to delirium caused by fever. The certificate should contain a clinical description of the patient, and indicate the reasons for the diagnosis of the specified disorder.

Feigned mental illness: Mental illness may be feigned by criminals to evade sentence of death or long terms of imprisonment, by soldiers and policemen to leave the service, and by businessmen to avoid contracts **(Table 22-3)**.

THE MENTAL HEALTH ACT, 1987: This act repeals the Indian Lunacy Act, 1912. The Act has changed the following terms: (a) Psychiatric hospital is used instead of mental hospital. (b) Mentally ill person instead of lunatic, unsoundness of mind instead of insane. (c) Mentally ill prisoner instead of criminal lunatic.

Table (22-3). Difference between real mental illness and feigned mental illness

Trait	Real mental illness	Feigned mental illness
(1) Onset:	Gradual.	Sudden.
(2) Motive:	Absent, e.g., no history of commission of crime.	Present, e.g., commission of crime.
(3) Predisposing factors:	Usually present, e.g., history of insanity in parents or of sudden monetary loss, grief, etc.	Absent.
(4) Signs and symptoms:	Uniform and present whether the patient is being observed or not.	Present only when conscious of being observed; variable and always exaggerated, and do not resemble any particular mental disease.
(5) Mood:	Excited, depressed or fluctuating.	May overact, to show abnormality in mood.
(6) Facial expression:	Peculiar, e.g., vacant look or fixed look of excitement.	No peculiarity; frequently changing, exaggerated and voluntary.
(7) Insomnia:	Present.	Cannot persist; patient sleeps soundly after a day or two.
(8) Exertion:	Patient can stand exertion of fatigue, hunger, and sleep for several days without breaking down.	Cannot stand exertion for more than a few days and breaks down.
(9) Habits:	Dirty and filthy.	Not dirty and filthy.
(10) Skin and lips:	Dry, harsh.	Normal.
(11) Frequent examination:	Does not mind.	Resents for fear of detection.

The main provisions of the Act are: (1) **"Mentally ill person" is defined as "a person who is in need of treatment by reason of any mental disorder other than mental retardation".** (2) The Central Government and State Governments have to establish an Authority for Mental Health, for regulation, development, and coordination with respect to Mental Health services. (3) Establishment and maintenance of psychiatric hospitals or psychiatric nursing homes can be done only with licence, which has to be renewed every five years. (4) If a mentally ill person is received or detained against the provisions of the Act, the punishment is imprisonment up to two years and also fine. (5) For every psychiatric hospital or nursing home, the Government should appoint not less than five visitors, of whom at least one should be a medical officer. Not less than three visitors shall make a joint inspection of hospital or nursing home, at least once in every month, and examine every minor patient, and as far as possible all other mentally ill persons, and the order for the admission of, and the medical certificates, and shall enter their remarks in a book.

RESTRAINT OF THE MENTALLY ILL PERSON

Restraint may be: (1) Immediate, and (2) Admission in psychiatric hospital.

(I) IMMEDIATE RESTRAINT: This can be done in case of: (1) a mentally ill person who is dangerous to himself or to others, or who is likely to wastefully spend his property or that of others, (2) person suffering from delirium due to disease, and (3) delirium tremens.

Immediate restraint is done under the personal care of attendants, e.g., by safely locking up in a room. The consent of the lawful guardian of the mentally ill person has to be taken, but if there is no time to take the consent, and the mentally ill person is dangerous to himself or others, he can be immediately restrained. Such restraint is lawful only as long as the danger exists.

(II) ADMISSION IN PSYCHIATRIC HOSPITAL: The following procedures are adopted.

(1) Admission on Voluntary Basis: Any major person, who considers himself to be a mentally ill person, may request the medical officer-in-charge of Psychiatric Hospital (or psychiatric nursing home), for admission and treatment. In case of a minor, the guardian may make such request. **The officer-in-charge should make such inquiry as he may deem fit within 24 hours, and if he is satisfied that the person requires treatment as an inpatient, he may admit such person.**

(2) Admission under Special Circumstances: A mentally ill person may not be able to express his willingness for admission as a voluntary patient. Such person can be admitted in a psychiatric hospital for a period of 90 days, if an application is made by a relative or friend. The application should be in the prescribed form and should be accompanied by two medical certificates, one of which shall be by a Government Medical Officer. If the application is not accompanied by medical certificates, the officer-in-charge of the psychiatric hospital, can get the mentally ill person examined by two doctors working in the hospital.

(3) Reception Order on Application: (A) The officer-in-charge of a psychiatric hospital can make an application to the Magistrate in case of a mentally ill person who is undergoing treatment under a temporary treatment order, if he is satisfied that (a) the treatment has to be continued for more than six months, or (b) it is necessary in the interest of the health and personal safety of the mentally ill person or for the protection of others.

(B) The husband or wife of the mentally ill person or any other relative can make an application in the prescribed form to the Magistrate. The applicant must be a major, and must have personally seen the patient within 14 days of the date of the application. The exact manner of relationship, and the circumstances under which the application had been made for inquiry should be stated. The application should be signed and verified by the applicant. Two medical certificates should be submitted, issued by two medical practitioners, who must have separately examined the patient within 10 days of the presentation of the application. One certificate should be from a Government Medical Officer. Each doctor should certify "that the alleged mentally ill person is suffering from mental disorder of such a nature and degree as to be detained in a psychiatric hospital, and that such detention is necessary in the interest of the health and personal safety of that person, or for the protection of others".

On receipt of application, the Magistrate must consider the allegations in the petition, and the evidence of mental illness appearing in the medical certificates. He can personally examine the alleged mentally ill person. If he is satisfied, he may pass a Reception Order immediately, or he may fix a date for consideration of the petition. In the latter case, he must give notice to the petitioner, and to any person to whom in his opinion, notice should be given. On the date fixed, the petition must be considered in private in the presence only of the petitioner, the alleged mentally ill person, a representative of the alleged mentally ill person, and such other persons as the Magistrate thinks should be present. If the Magistrate is satisfied that it is necessary to detain the alleged mentally ill person in a psychiatric hospital, he passes a **Reception Order (order for admission and detention) which is valid for 30 days.** If he is not satisfied, he may refuse the application, giving his reasons in writing, a copy of which is supplied to the applicant. A certified copy of the Reception Order is sent to the officer-in-charge of the psychiatric hospital.

(4) Reception Order on Production of Mentally Ill Person before Magistrate: (a) An officer-in-charge of a police station is authorised to arrest a **wandering or dangerous mentally ill person,** and produce him before a Magistrate. A wandering mentally ill person is one who wanders aimlessly, and a dangerous mentally ill person is one who because of his violent behaviour is dangerous to himself or others. The Magistrate must examine such person, send him to be examined by a medical officer, and make necessary inquiries, and if he is satisfied that such person is mentally ill, he may pass a Reception Order. If a relative or friend executes a bond for the proper care of the mentally ill person, the Magistrate may hand him over to the friend or relative.

(b) An officer-in-charge of a police station, or any private person can report to a Magistrate, if he believes that any person is mentally ill, and is not kept under proper care and control, or that he is **cruelly treated or neglected** by any relative or guardian. The Magistrate may order to produce the mentally ill person before him, and summon such relative or guardian. The Magistrate may make an order for the proper care and treatment of the mentally ill person. If there is no one who is legally bound to maintain the mentally ill person and even otherwise, the Magistrate may pass an order for the admission of the mentally ill person.

(5) Admission after Judicial Inquisition: If a person possessing property is alleged to be mentally ill, the District Court may order an inquisition upon application made by any relative. The Court, if it is satisfied, may order to admit such person in a psychiatric hospital.

(6) Admission of Mentally Ill Prisoner: A mentally ill prisoner can be admitted into any psychiatric hospital, by an order passed by an Appropriate Authority under Prisoners Act (1900), Air Force Act (1950), Army Act (1950), Navy Act (1957), or under Section 330 or 335 of Cr.P.C. (1973).

(7) Admission of an Escaped Mentally Ill Person: A mentally ill person escaping from a psychiatric hospital, can be retaken by any police officer, or any officer or servant of the psychiatric hospital and readmitted into such hospital.

Discharge of a Mentally Ill Person: (1) Voluntary patients should be discharged within 24 hours of the receipt of request for discharge made by the patient or the guardian. (2) A mentally ill person who is admitted on an application by a relative or friend can himself, or a relative or friend can apply to the Magistrate for his discharge. The Magistrate after making an enquiry can either allow or dismiss the application. (3) The officer-in-charge of a psychiatric hospital can order in writing discharge of any inpatient on the recommendation of two medical practitioners, one of whom shall preferably be a psychiatrist. (4) A mentally ill person detained under Reception Order made on application, shall be discharged, if the person on whose application the admission order was made, applies in writing to the officer-in-charge. (5) If a person detained on Reception Order, is subsequently found by any judicial inquisition to be of sound mind, he is to be discharged.

MENTAL DISORDER AND RESPONSIBILITY

Responsibility, in the legal sense means the liability of a person for his acts or omissions, and if these are against the law, the liability to be punished for them. The law presumes that every person is mentally sound until disproved.

CIVIL RESPONSIBILITY

The question of civil responsibility arises in the following conditions:

(1) Management of Property and Affairs of Mentally ill Person: If any relative or friend of an alleged mentally ill person possessing property gives an application, the Court may direct an enquiry. The medical evidence is given in the form of a certificate, which should state "that mental illness is of such a degree as to make him incapable of managing his property and affairs". If on enquiry, the person is found incapable of managing his property and affairs, but is not dangerous to himself or to others, the Court appoints a manager to look after his property, granting him necessary powers. The Court may order the sale or disposal of the property of the mentally ill person for the payment of his debts and expenses. The Court may order a second inquiry, if it is reported that unsoundness of mind had ceased, and order all proceedings in the unsoundness of mind to cease, if it is satisfied that the unsoundness of mind has ceased.

(2) Mental illness and Contracts: A contract is invalid, if one of the parties at the time of making it was incapable of understanding what he was doing due to unsoundness of mind. Contract entered into with a mentally ill person may be valid, if the other party can show that he did not know that the other party was mentally ill, and the contract is a fair contract. Mental illness which develops subsequent to the contract does not make it invalid, unless performance of services becomes impossible. A mentally ill person is responsible for the payment of the simple necessities of life, such as food, shelter, clothing, medical care, etc., but he is not responsible if the order is grossly excessive or unreasonable, or

if the seller has taken undue advantage of his mental illness.

(3) Mental illness and Marriage Contract: A marriage is considered invalid if at the time of marriage, either party (1) is incapable of giving valid consent due to mental illness, or (2) though capable of giving valid consent, has been suffering from such a kind or degree of mental disorder as to be unfit for marriage and procreation, or (3) has been suffering from recurrent attacks of mental illness or epilepsy.

(4) The Competence of Mentally ill Person to be a Witness: A mentally ill person is not competent to give evidence, if he cannot understand the necessity of telling the truth due to mental illness. A person of unsound mind who suffers from delusions, but is capable of telling of what he has seen, and who understands the importance of an oath, is competent to give evidence. A mentally ill person is competent to give evidence during the period of lucid interval.

(5) Consent and Mental Illness: Consent to certain acts, like sexual intercourse or hurt is not valid, if such consent is given by a person who from unsoundness of mind is unable to understand the nature and consequences of that act.

(6) MENTAL ILLNESS AND TESTA MENTARY CAPACITY: Testamentary capacity (testament = will) is **the mental ability of a person to make a valid will.**

Requirements: The requirements for a valid will are as follows: A written and properly signed and witnessed document must exist. The person making the will must be major, of sound disposing mind, and at the time of making the will, force, undue influence, or dishonest representation of facts should not have been applied by others. **Holograph will** is one which is **written by a testator in his own handwriting.**

Medical examination: Doctors are sometimes called upon to witness the execution of the will of a sick person. The doctor should proceed in the usual way: physical examination, mental state including intelligence testing, and laboratory investigations. The testator is said to be of sound mind if he is capable of disposing of his property with understanding and reason. **The following tests are recommended to find out whether the testator is of sound and disposing mind.** (1) Ask the testator preliminary questions, e.g., about his relatives, their number and the degree of social contact with them, his opinions on family, friends and business partners, his age, politics and hobbies. (2) Ask general questions for testing awareness as regards time, place, etc. (3) Ask him about the nature, extent and value of his properties, and the manner of distribution desired by him. If any unusual or unjust distribution is to be made, find out whether it is intentional and if so the reasons for it, and whether he is able to repeat the main provisions of will. (4) Test the patient's powers of concentration by simple sums of arithmetic, etc. (5) Ask all other persons to leave the room, and then ask the patient whether there was any pressure or influence on him by any one. The doctor should exclude any disease, physical weakness, pain, strain, influence of drug or drink or any delusion, due to mental illness, which are bound to affect a normally sound and disposing state of mind. **The most common symptom of absence of legal capacity is impairment of memory. The most important thing to determine is whether at the time of making the will, the testator understood the business in which he was engaged, and knew how he wanted to dispose off his property.**

Valid will: (1) A person affected by delusion due to mental illness can make a valid will, if the delusion is not related in any way to disposal of the property or the persons affected by the will. (2) Persons can make valid wills during lucid interval. (3) Persons of extreme age and feeble health with defective memory, can make a valid will unless their mind has become so impaired that they are unable to understand its nature and consequences. (4) A person suffering from motor or sensory aphasia, agraphia (failure to communicate by writing) and alexia (failure to understand by reading) or who is deaf, dumb or blind can make a valid will, if he knows what he does by it, if he can make clear by gestures that he wishes to make a will, and is able to understand the meaning of questions put to him in this connection.

Invalid will: (1) Wills made by persons at extreme age (at the point of death) may be regarded with suspicion, because at that time a clear mind is unusual. (2) A will executed by a dying person during delirium would be invalid. (3) Partial drunkenness does not invalidate a contract or will, but when drunkenness

has caused a temporary loss of reasoning powers, the person cannot make a valid will.

An executor(s) is appointed under the will by the testator to carry out terms after his death.

CRIMINAL RESPONSIBILITY

A person may plead mental illness to avoid (1) trial, when the accused is mentally ill and cannot plead, (2) conviction, if the accused was mentally ill when the alleged crime was committed, (3) sentence of capital punishment, when a condemned prisoner is mentally ill. If mental illness is proved, the accused person is found "not guilty" and is ordered to be kept in psychiatric hospital, or other suitable place of custody. **The law presumes that every person is sane and responsible for his actions. The defence has to prove that the accused is of unsound mind. The law also presumes that for every criminal act, there must be criminal intent or mind, mens rea** (mens = mind; rea = criminal) **motivating it. Actus reus means the actual forbidden physical act causing death. Tests for determining criminal responsibility.**

(1) MC NAUGHTEN RULE (The right or wrong test; the legal test): Daniel Mc Naughten was a paranoid schizophrenic, who had the delusional belief that he was being persecuted, and this could only be stopped by shooting Sir Robert Peel, at that time Prime Minister of England in 1843. He attempted to do so, but by mistake shot and killed Edward Drummond, the Prime Minister's private secretary. Mc Naughten was acquitted as a result of medical evidence which showed him to be of unsound mind. He spent the remaining period of his life in psychiatric hospital. The public reaction to his acquittal was most unfavourable, and lead to debate in the House of Lords. A number of questions were put to full bench of fourteen Judges. The answers have become known as the Mc Naughten Rules. **"An accused person is not criminally responsible, if it is proved that at the time of committing the crime, he was suffering from such a defect of reason from abnormality of mind, that he did not know the nature and quality of the act he was doing, or that what he was doing was wrong." This legal test has also been accepted in India as the law of criminal responsibility, under Sec. 84, I.P.C.** Nothing is an offence, which is done by a person, who at the time of doing it by reason of unsoundness of mind, is incapable of knowing the nature of the act or that he is doing what is either wrong or contrary to law."

Examples: (1) If a mentally ill person has a delusion and thinks that another man is attempting to kill him, and he kills that man in self-defence, he has no criminal responsibility. (2) If under the influence of delusion due to mental illness a person thinks another to be a wild animal and kills him, he has no criminal responsibility, because he does not know the physical nature of the act. (3) If under the influence of delusion due to mental illness a person thinks that he is the state executioner and that he has to execute the victim as a part of his job, he has no criminal responsibility. (4) If under the influence of delusion due to mental illness a person thinks that another person has caused a serious injury to his character and fortune and kills him, he becomes responsible, because under the law no one can kill a person in revenge.

The defect of Mc Naughten rule is, that for deciding that a person is mentally ill, only intellectual factors (reason) are taken into consideration, but not the emotional factors, delusional beliefs, hallucinations and the ability of the individual to control the impulses.

(2) Durham Rule (1954). (3) Curren's Rule (1961). (4) The Irresistible Impulse Test. (5) The American law Institute Test (A.L.T.).

MENTAL ILLNESS AND MURDER: Medical examination: In criminal cases where mental illness is pleaded as a defence, the defence has to prove it. The doctor should obtain detailed history from the accused person and from other sources, and then carry out physical examination and investigations. The following factors which are helpful, should be taken note of: **(1) History:** History of the accused and his family with regard to mental disease. **(2) Motive:** There is no motive in mental illness. **(3) Preparation:** Prearrangement or preplanning are absent in mental illness. **(4) Accomplices:** Not present. **(5) Nature of crime:** A person may kill several persons including his friends and relatives. **(6) Conduct of the criminal at the time of the crime:** A mentally ill person does not try to destroy evidence. **(7) Conduct of the criminal after the crime:** A mentally ill person may even notify police about the crime.

Mental state of the alleged criminal: The law considers the state of mind, only at the time of the alleged offence and not generally. **The mental status of responsibility at the time of the alleged offence has to be determined.** The capacity to stand trial is determined at the time of the actual trial. The interval between these two events may be days, months or years.

Psychiatrist's opinion: The psychiatrist conducts the examination of the alleged mentally ill person at some time after the alleged crime, and before the trial. The psychiatrist's task becomes difficult with the increase in the interval between the alleged crime and his examination. Psychiatric prediction has limited reliability. An opinion of the present mental state of an individual usually carries the lowest risk of error, an opinion about a recent past mental state has greater risk of error, and an opinion about the future carries the greatest error and least reliability. **The greater the interval between commission of crime and psychiatric examination, the lower will be the reliability of the opinion for legal purposes.** The opinion of the psychiatrist about the mental state of an accused at a previous period of time depends on his knowledge of the accused's present clinical condition, past history obtained from relatives and medical sources, his behaviour before, during and after the act in question, and his knowledge about the course and natural history of the disorder of mind. Therefore, at the very best, the psychiatrist's views concerning the accused's degree of responsibility and self-control at the time of alleged offence will be matters of inference. **If he finds evidence of frank psychosis, epilepsy or other illness accepted as disease or disorder of mind, he can justifiably infer that such illness played a part in the offence with which the accused is charged.**

Legal considerations: All the mental disorders do not free a person from criminal responsibility for his acts. **If the disorders impair the cognitive faculties (all aspects of perceiving, thinking and remembering) of the accused, i.e., the faculty of understanding the nature of his act and its consequences, he is not held responsible. If mental illness affects only the emotions and the will, but not the cognitive faculties, the person is held responsible for his acts. The law recognises a guilty intention, the mens rea, as an essential part of the crime. Only those persons who are completely incompetent, demented or wild, are considered as lacking the ability to have a guilty intention.** When a medical witness deposes regarding the existence, characters, and extent of the mental disease, the Judge has to decide whether the disease justifies an acquittal on the ground of insanity.

Doctrine of diminished responsibility: The term is used for borderline mental state. It recognises that there are different degrees of mental disorder, and in general a defence of diminished responsibility requires evidence of a state of mind bordering upon but not amounting to insanity. This includes certain organic states, depression, obsessional states and some paranoid states. The defence of diminished responsibility is usually applied for people with a lesser degree of mental abnormality (whether arising from a condition of arrested development or any inherent causes or induced by disease or injury) that could be brought within the Mc Naughten rules. **Such person may be punished for culpable homicide not amounting to murder.**

AUTOMATISM: Automatism is conduct of a person whose consciousness is impaired to such an extent that he is not fully aware of his actions. There may be no consciousness at all of the actions in question, or there may be awareness that falls below the level of normal consciousness. This alteration of consciousness may be produced by organic factors or by non-organic factors, such as stress or shock. **The main factors producing automatism recognised by the Criminal Courts are: (1) Epilepsy (mentally ill automatism). (2) Concussion or cerebral disease. (3) Hypoglycaemia, and (4) Somnambulism (non-mentally ill automatism).** The concept of automatism is very vague and unsatisfactory. The Indian law has no special provision for automatism.

SOMNAMBULISM: It means, **walking during sleep. It is characterised by aimless wandering with incomplete arousal from sleep, attended with acute anxiety.** A person leaves his bed and walks in the house or out of the house without any awareness of his actions, but rarely injures himself. He is not asleep but in a state of dissociated consciousness, in a hallucinatory state, unrelated to his immediate environment. **It is similar to an automatism.** Such persons are usually well-adjusted in life, socially

well-behaved, and not aggressive. A large percentage of adults have psychiatric problems. There may have been immediate stress or concealed mental conflict before the walk. The crime is not wilful or premeditated. The mental faculties are partially active and are so concentrated on one particular idea, that he may solve a difficult problem or commit a crime, e.g. theft or murder. **He may commit suicide, fall in a well, meet with an accident or commit a crime. There is no recollection of the event, but in some cases, the events of one fit are remembered in a subsequent fit and carried out similarly. Such person is not criminally responsible for his acts.**

SOMNOLENTIA (SEMISOMNOLENCE): It is often **called sleep-drunkenness and is midway between sleep and waking.** If such person is suddenly aroused from a deep sleep, he may commit some crime, due to confusion of the mind, especially when he is having a dream at that time. **Such person is not criminally responsible for his acts.**

Impulse: Some crimes are committed due to an impulse, in which the person loses self-control, such as sudden violent anger, etc. **Such persons are criminally responsible unless mental illness is present.**

Hypnotism or Mesmerism: This is a sleep-like condition produced by artificial means or by suggestions. During a hypnotic trance, a person may perform acts suggested by the hypnotiser, but does not remember them afterwards. **A hypnotised person usually cannot be tricked into doing some immoral or dishonest act.**

Delirium: A delirious person may commit violent criminal acts due to delusions and hallucinations. **He is not legally responsible for the acts committed during delirium.**

Drunkenness: An act done by a person who is incapable of knowing the nature of the act due to intoxication is not an offence, if the thing which intoxicated him was administered to him without his knowledge or against his will (S. 85, I.P.C.). An intoxicated person is criminally responsible if he has the intention or knowledge of committing a crime **(S. 86, I.P.C.).** Thus, if a drunken person commits criminal abortion due to which the woman dies, the abortionist may be guilty of murder, for he intended to commit an unlawful act. **Mental disorder brought about by drugs, and delirium tremens due to drink, frees one of criminal responsibility.**

A person is **not responsible** for his criminal **acts done during post-traumatic automatism, twilight state and oneiroid states.**

CHAPTER

23 General Considerations

Toxicology is the science dealing with properties, actions, toxicity, fatal dose, detection and estimation of, interpretation of the result of toxicological analysis, and treatment of poisons. **Forensic toxicology deals with the medical and legal aspects of the harmful effects of chemicals on human beings.** A **poison is a substance (solid, liquid or gaseous) which if introduced in the living body, or brought into contact with any part thereof, will produce ill-health or death, by its constitutional or local effects or both.**

HISTORY: References to poisons are found in the oldest Egyptian, Babylonian, Hebrew and Greek records. The ancient Indian Scriptures contain references to the poisoning of kings, and of widespread organised poisoning by professional poisoners in almost prehistoric times. In Kalpasthana, Chikitsasthana, and Uttarasthana of the Shastras, symptoms and antidotes of poisons are given in detail. Susruta described several modes of poisoning in ancient India.

THE LAW OF POISONS

(1) POISONS ACT, 1919: It regulates the importation, possession and sale of poisons.

(2) THE DRUGS AND COSMETICS ACT, 1940: It regulates the import, manufacture, distribution and sale of all kinds of drugs. One of its main features is the control of the quality, purity and strength of drugs. This Act empowers the Central Government to form a Drugs Technical Advisory Board, and to establish a Central Drugs Laboratory, to help and advice both the Central and State Governments.

(3) THE DRUGS AND COSMETIC RULES, 1945: They were framed under the Drugs Act, 1940, to regulate the importation of drugs, the functions and procedures of the Central Drugs Laboratory, the appointment of licensing authorities, and the manufacture, distribution and sale of drugs. These rules have classified drugs into schedules. Schedule H: Drugs (Poisons to be sold only on the prescription of a registered medical practitioner).

(4) THE PHARMACY ACT, 1948: It was passed to make better provision for the regulation of the profession of pharmacy and to constitute Central and State Councils of Pharmacy.

(5) THE DRUGS CONTROL ACT, 1950: It provides for control of sale, supply and distribution of drugs. It gives power to fix the maximum price of any drug, which may be charged by a dealer.

(6) THE DRUGS AND MAGIC REMEDIES (OBJECTIONABLE ADVERTISEMENT) ACT, 1954: The object is to ban advertisements which offend decency or morality and to prevent self-medication and treatment which cause harmful effects.

(7) NARCOTIC DRUGS AND PSYCHOTROPIC SUBSTANCES ACT, 1985 (AMENDED IN 1989 AND 2001): A psychotropic drug is one that alters mental function by its action. A narcotic drug means Cocoa leaf, Cannabis, Opium, Poppy straw and includes all manufactured drugs. "Psychotropic substance" means any substance, natural or synthetic, or any natural material or any salt or preparation of such substance, or material, included in the list of psychotropic substances specified in a schedule to the Act. This schedule lists 77 psychotropic substances, e.g., LSD, amphetamine, tranquilisers, barbiturates, etc.

(8) PREVENTION OF ILLICIT TRAFFIC IN NARCOTIC DRUGS AND PSYCHOTROPIC SUBSTANCES ACT, 1988: If a person produces, possesses, transports, imports, exports, sells, purchases or uses any narcotic drug or psychotropic substance except ganja, he shall be punishable with imprisonment.

Medicolegal Aspects of Poisons: Sections 176, 177, 193, 201, 202, 284, 299, 300, 304 A, 309, 320, 324, 326 and 328 I.P.C. deal with offences relating to administration of poisonous substances. **The intention with which any act is committed is an important element in law. The law does not make any difference between murder by means of poisons, and murder by other means.** Deliberate administration of poison proves the intention of the accused to cause death, or other such bodily injury which is sufficient to cause death, and will bring culpable homicide within the scope of murder.

Poisoning in India: Suicidal and homicidal cases of poisoning are common in India, as poisons can

be easily obtained and many poisonous plants grow wild, e.g., datura, oleanders, aconite, nux vomica, etc. Accidental poisoning occurs from use of philters or love potions and quack remedies containing poisonous drugs and snake bites. A love philter is a drug which is supposed to increase the love between the giver and taker. All aphrodisiacs such as cantharides, arsenic, alcohol, opium, cocaine and cannabis are supposed to act as love philters.

NATURE OF POISONING

(1) Ideal Homicidal Poison: The characters of an ideal homicidal poison should be: (1) cheap, (2) easily available, (3) colourless, odourless and tasteless, (4) capable of being administered either in food, drink or medicine, without producing any visible change, (5) highly toxic, (6) signs and symptoms should resemble a natural disease, or the serious ill-effects should be delayed sufficiently long for the criminal to escape suspicion, (7) there should not be any antidote, (8) there should be no postmortem changes, (9) should not be detected by chemical tests, or other methods, and (10) must be rapidly destroyed in the body. **Organic compounds of fluorine (used as rodenticides) and thallium satisfy several of the above criteria. Arsenic and aconite are commonly used as homicidal poisons.**

(2) Ideal Suicidal Poison: The characters of an ideal suicidal poison should be: (1) cheap, (2) easily available, (3) highly toxic, (4) tasteless or of pleasant taste, (5) capable of being easily taken in food or drink, and (6) capable of producing painless death. **Opium and barbiturates satisfy several of the above criteria. Organophosphorus compounds, chlorinated hydrocarbons, aluminium phosphide, carbamates, pyrethroids and endrin are commonly used as suicidal poisons.**

(3) Stupefying: Datura, cannabis indica, chloral hydras.

(4) Abortion: Calotropis, oleanders, aconite, ergot, croton, semecarpus, lead, arsenic, mercury, potassium permanganate, cantharides, etc.

(5) Accidental: Common household poisons (**Table 23-1**).

(6) Rare: Bacteria, insulin.

(7) Non-accidental poisoning may occur in cases of child abuse, usually below 30 months.

(8) Cattle Poisoning: The usual motive is destruction of cattle of an enemy or to obtain the hides. The usual poisons are abrus precatorius, oleanders, calotropis, organophosphorus, arsenic, aconite, strychnine, zinc phosphide, nitrate, etc.

(9) Arrow Poisons: Abrus precatorius, croton oil, calotropis, aconite, strychnine, curare and snake venom.

(10) Aphrodisiacs: Cantharides, cocaine, cannabis, opium, strychnine, arsenic.

Classification: Poisons may be classified according to the chief **symptoms** which they produce.

(I) Corrosives: (1) Strong acids: (a) **Mineral or inorganic acids:** Sulphuric, nitric, hydrochloric, chromic, boric, phosphoric. (b) **Organic acids:** Carbolic, oxalic, acetic, salicylic, citric, formic. **(2) Strong alkalis:** Hydrates and carbonates of sodium, potassium and ammonia. **(3) Metallic salts:** Zinc chloride, ferric chloride, copper sulfate, silver nitrate, potassium cyanide, chromates, bichromates.

(II) Irritants: (1) Agricultural. (2) Inorganic: (a) **Non-metallic:** Phosphorus, iodine, chlorine, bromine, carbontetrachloride. (b) **Metallic:** Arsenic, antimony, copper, lead, mercury, silver, zinc, (c) **Mechanical:** Powdered glass, diamond dust, hair, etc. **(3) Organic:** (a) **Vegetable:** Abrus precatorius, castor, croton, calotropis, aloes. (b) **Animal:** Snake and insect bites, cantharides, ptomaines.

(III) Systemic: (1) Cerebral: (a) **CNS depressants:** Alcohols, general anesthetics, opioid analgesics, hypnotics, sedatives. (b) **CNS stimulants:** Cyclic antidepressants, amphetamine, methylphenidate, caffeine. (c) **Deliriant:** Datura, belladonna, hyoscyamus, cannabis, etc. (2) **Spinal:** Nux vomica, gelsemium. (3) **Peripheral:** Conium, curare. (4) **Cardiovascular:** Aconite, quinine, oleanders, tobacco. (5) **Asphyxiants:** CO, CO_2, hydrogen sulphide, cyanides.

(IV) Miscellaneous: Food poisoning, botulism.

Poisoning may result from: (1) The administration of a poison for criminal purposes. (2) The swallowing of poison in mistake for harmless substance. (3) The inhalation through ignorance or accident of the vapours of a poison. (4) The incorrect preparation of medicines containing a poison. (5) The accidental taking of a large dose of medicine containing a poison.

Table (23-1). Common household poisons

Preparation	Toxic substance
(A) DOMESTIC POISONS:	
(I) Cosmetics:	
(1) Cuticle remover:	Potassium hydroxide; trisodium phosphate.
(2) Depilatories:	Barium sulphide; thallium.
(3) Hairwave lotions:	Thioglycollate salts; perborates; bromates.
(4) Nail polish removers:	Acetone.
(5) Suntan lotions:	Denatured alcohol; methyl salicylate.
(6) Baby powder:	Boric acid.
(II) Kitchen:	
(1) Baking powder:	Tartaric acid 50%.
(2) Baking soda:	Sodium bicarbonate.
(3) Dish washing compounds:	Sodium polyphosphates; sodium carbonate; sodium silicates.
(4) Fire extinguishing fluids:	Carbontetrachloride; sodium carbonate; methyl bromide.
(5) Matches:	Antimony; red phosphorus; sesqui-sulphide; potassium chlorate.
(III) Rat poisons:	
(1) Rat paste:	Aluminium phosphide; zinc sulphide; zinc phosphide; arsenious oxide; red squill, thallium sulphate; phosphorus; barium carbonate.
(2) Rodine	Yellow phosphorus.
(IV) Roach powder:	Sodium fluoride.
(V) Sanitary:	
(1) Deodorant tablets:	Formaldehyde; naphthalene.
(2) Drain cleaners:	Sodium hydroxide.
(3) Disinfectants:	Phenol: bleaching powder.
(VI) Miscellaneous:	
(1) Insecticide spray:	D.D.T., Gammexane, etc.
(2) Moth balls:	Naphthalene.
(3) Marking ink:	Aniline.
(4) Ink remover:	Sodium hypochlorite 5%.
(5) Anti-rust products:	Ammonium sulphide, hydrofluoric acid, naphtha, oxalic acid.
(6) Cleaning solvents:	Petroleum hydrocarbons; carbon tetrachloride; trichloroethylene.
(7) Fluorescent lamps:	Beryllium.
(8) Furniture polish:	Turpentine; petroleum hydrocarbons.
(9) Paint remover:	Sodium hydroxide; lead acetate.
(10) Shoe polish:	Aniline; nitrobenzene.
(11) Hair bleach:	Potassium permanganate: hydrogen peroxide.
(12) Toys (paints):	Lead.
(13) Fireworks:	Arsenic, mercury; antimony; lead; phosphorus; thiocyanate.
(14) Crayons (chalk):	Salts of arsenic, copper, lead.
(15) Crayons (wax):	Paranitroaniline.
(B) GARDEN POISONS:	
(1) Insecticides:	Organophosphorus compounds; chlorinated hydrocarbons; nicotine: tar oils.
(2) Fungicides:	Lead arsenate: copper compounds; organic mercurials; lime; sulphur.
(3) Weed-killers:	Sodium chlorate; arsenious oxide, and arsenites; dinitrocresol; paraquat.
(C) THERAPEUTIC POISONS:	
(1) Antiseptics:	Iodine, benzoin.
(2) Tonic tablets:	Iron.
(3) Tonic syrup:	Strychnine.
(4) Sleeping tablets:	Barbiturates.
(5) Headache tablets:	Aspirin.
(6) Cough remedies:	Codeine.
(7) Throat tablets:	Potassium chlorate.
(8) Pep tablets:	Benezedrine.
(9) Others:	Antidepressants; tranquilisers.

(6) Excessive self-medication. (7) Addiction to drugs. (8) Bite by a poisonous animal. (9) Food infected with bacteria or their toxins.

Routes of Administration: In order of rapidity of action: (1) Inhaled in gaseous or vaporous form. (2) Injection into blood vessels. (3) Intramuscular, subcutaneous and intradermal injection. (4) Application to a wound. (5) Application to a serous surface. (6) Application to a bronchotracheal mucous membrane. (7) Introduction into stomach. (8) Introduction into the natural orifices, e.g., rectum, vagina, urethra, etc. (9) Application to unbroken skin. Organic phosphates, nicotine, insecticides, some organic solvents and lewisite gas can penetrate the skin and produce death. Other substances which are absorbed through the skin are: phenol and its derivatives, endrin, methyl salicylate, mercury, tetraethyl lead, cantharidin, hydrocyanic acid, oestrogen, progesterone, desoxycorticosterone, vitamin D and K.

FATE OF POISONS IN THE BODY: The greater part of a poison is thrown out of the body as a result of vomiting and purging. The portion absorbed is mainly deposited in a less soluble form in the liver which metabolises or completely destroys it. The unaltered portion enters into the general circulation and acts on the particular organ with which it has special affinity, provided the poison is not destroyed or made harmless by the kidneys and muscles. Some inorganic poisons like arsenic and antimony are retained in certain tissues, such as nails, hair, bones, etc., for a considerable time. Certain poisons like chloroform, phosphorus, nitrates, aconite and acetic acid disappear by evaporation or oxidised or destroyed in the body and no trace of them can be detected in the viscera or tissues if postmortem is delayed.

ROUTES OF ELIMINATION: The absorbed portion of poison is mainly excreted by the kidneys, and to some extent by the skin. Other routes are bile, milk, saliva, mucous and serous secretions. The unabsorbed portion is excreted in the vomit and faeces.

Pharmacodynamics is the action of drugs on living organisms. **Pharmacokinetics** is the study of action of a drug in the body over a period of time including processes of absorption, distribution, localisation in tissues, biotransformation and excretion.

ACTION OF POISONS: (1) LOCAL: The local action by coming in direct contact with the part. (1) Chemical destruction by corrosives. (2) Congestion and inflammation by irritants. (3) Effects on motor and sensory nerves, e.g., tingling of skin and tongue by aconite, dilation of pupils by belladonna or datura.

(2) REMOTE: Remote action is produced either by shock acting reflexly through severe pain caused by corrosive or by poison being first absorbed into the system through the blood, and then exerting a specific action on certain organs and tissues, e.g., cantharides acting on kidneys produces nephritis, nux vomica acting on the spinal cord produces convulsions.

(3) COMBINED: Drugs like carbolic acid, oxalic acid, phosphorus, etc., have local and remote actions.

CAUSES MODIFYING ACTION OF POISONS

(1) QUANTITY: A large quantity of poison taken orally may cause excessive vomiting, causing its rapid elimination and decreased toxicity, e.g., alcohol, copper sulfate, etc. The action of a poison varies with the dose, e.g., a very large dose of arsenic may produce death by shock without causing irritant symptoms, while moderate doses produce irritant symptoms, and small doses produce therapeutic action (Table 23-2).

(2) FORM: (a) PHYSICAL STATE: Poisons act most rapidly when gaseous, and less when liquid. In case of solids, the action depends on their solubility. (b) CHEMICAL COMBINATION: The action of a poison depends upon the solubility or insolubility resulting from a chemical combination, e.g., silver nitrate and hydrochloric acid are both strong poisons, but when combined, form an insoluble salt of silver chloride which is harmless. A substance may be harmless in metallic state but its salt may be toxic, e.g., arsenic is not poisonous, but its salts are poisonous. Certain poisons which are not soluble in water may become dissolved in the acid secretion of the stomach and absorbed into the blood, e.g., lead carbonate and copper arsenite. (c) MECHANICAL COMBINATION: The action of a poison may be altered if combined

Table (23-2). Toxicity rating (Gosselin, et al.)

Usual fatal dose	Rating
<5 mg/kg	6 (super toxic)
5 to 50 mg/kg	5 (extremely toxic)
50 to 500 mg/kg	4 (very toxic)
0.5 to 5 g/kg	3 (moderately toxic)
0.5 to 15 g/kg	2 (slightly toxic)
>15 g/kg	1 (non-toxic)

mechanically with inert substances, e.g., small dose of concentrated mineral acid produces corrosive action, but the same dose largely diluted with water is harmless.

(3) MODE OF ADMINISTRATION: The rapidity of the action is in the order described under routes of administration. As a rough guide, if the active dose by the mouth is considered as unit, the rectal dose is about one-and-half to two, and the hypodermic dose about one-fourth. A lethal dose is usually 10 or more times the maximum medicinal dose.

(4) CONDITION OF THE BODY: (a) AGE: Poisons have greater effect at the two extremes of age. (b) IDIOSYNCRACY: Certain people are sensitive for certain drugs and even articles of diet, e.g., shellfish, eggs and fruit. Iodine, bromine, opium, heroin, cocaine, penicillin, belladonna, aspirin and mercury are common examples of drugs to which many people are allergic. (c) HABIT: The effect of certain poisons decrease with habituation, such as opium, alcohol, tobacco, etc. (d) STATE OF HEALTH: A healthy person tolerates better than a diseased. General debility, senility, chronic or disabling disease may cause death of a person to a dose that is ordinarily safe. (e) SLEEP AND INTOXICATION: The action of a poison is delayed if a person goes to sleep soon after taking it. The action is also delayed if one takes a poison in an intoxicated condition. (f) CUMULATIVE ACTION: Poisons which are eliminated slowly may accumulate in the body when given in repeated doses for a long time and may ultimately produce symptoms of poisoning.

Types of Poisoning: (1) Fulminant poisoning is produced by a massive dose. In this death occurs rapidly, sometimes without preceding symptoms. (2) Acute poisoning is caused by an excessive single dose or several smaller doses taken over a short interval of time. (3) Subacute poisoning shows features of both acute and chronic poisoning. (4) Chronic poisoning is caused by smaller doses over a period of time, resulting in gradual worsening. The poisons which are commonly used for the purpose of chronic poisoning are: arsenic, phosphorus, antimony and opium.

Self-poisoning (attempted suicide, parasuicide or pseudocide) is a conscious, often impulsive, manipulative act, undertaken to rectify an intolerable situation. Most persons are psychologically disturbed.

EPIDEMIOLOGY: Poisoning both accidental and intentional are a significant contributor to mortality and morbidity throughout the world.

The exact incidence of poisoning in India is uncertain, due to lack of data at central level, as most cases are not reported, and as mortality data are a poor indicator of incidence of poisoning. It has been estimated that about 5 to 6 persons per lakh population die due to poisoning every year.

In India, the common poisons are insecticides and pesticides, such as organophosphates, chlorinated hydrocarbons, aluminium phosphide, carbamates and pyrethroids. Other poisons are corrosives, sedatives, alcohol, datura, oleanders, calotropis, croton and cleaning agents. The reasons being agriculture based economics, poverty and easy availability of highly toxic pesticides. In children, kerosene, pesticides, drugs and household chemicals are commonly involved. Occupational poisoning due to pesticides are also common in developing countries, due to unsafe practices, illiteracy, ignorance and lack of protective clothing. In India, organophosphates form the largest bulk of pesticide poisoning. Since 1985, aluminium phosphide poisoning has been reported as the commonest cause of intentional poisoning in northern parts of India, viz., Haryana, Punjab and Rajasthan.

ECOTOXICOLOGY: It is concerned with the toxic effects of chemical and physical agents on living organisms, especially in populations and communities within defined ecosystems. It includes the transfer pathways of those agents and their interactions with the environment.

Poison information centres: National Poisons Information Center has been established in AIIMS, New Delhi. It uses a computer software on poisons (INTOX) compiled by WHO. National Institute of Occupation Health at Ahmedabad has also a center. Regional centers are located in Chennai and Cochin (POISINDEX). These centers provide toxicity assessment and treatment recommendations over the telephone throughout the day for all kinds of poisons.

PREVENTION: (1) Education is a major component of any poison prevention programme. (2) All drugs and toxic substances should be kept in locked cabinets. (3) All household poisons must be kept separate from food. (4) All pesticides and medicines should be kept in their original containers. (5) Keep caps and tops on bottles properly closed. (6) Do not keep household cleaners on the floor, under the kitchen sink, or in low cupboards that a child can easily open. (7) The label should be read before using the drug. (8) No drug should be given or taken in the dark. (9) All drugs

whether expired or otherwise should be disposed in a safe manner. (10) Drugs in child-proof packages only should be purchased. (11) Children should be taught not to eat plants or berries. (12) Wherever cooking gas is used, adequate ventilation should be provided. (13) In persons showing suicidal tendencies, special care should be taken. (14) In chemical factories air pollution should be prevented. (15) The workers in all factories should be properly educated, and safety equipment provided.

DIAGNOSIS OF POISONING IN THE LIVING: There is no single symptom, and no definite group of symptoms, which are absolutely characteristic of poisoning. The following conditions should arouse suspicion of poisoning. (1) The symptoms appear suddenly in a healthy person. (2) The symptoms appear immediately or within a short period after food or drink. (3) The symptoms are uniform in character, and rapidly increase in severity. (4) When several persons eat or drink at the same time from the same source of poison, all suffer from similar symptoms at or about the same time. (5) The detection of poison in food taken, in the vomit or in the excreta is strong proof of poisoning.

Symptoms: The following groups of symptoms are suggestive of poisoning. (1) The sudden onset of abdominal pain, nausea, vomiting, diarrhoea, and collapse. (2) The sudden onset of coma with constriction of pupils. (3) The sudden onset of convulsions. (4) Delirium with dilated pupils. (5) Jaundice and hepatocellular failure. (6) Oliguria with proteinuria and haematuria. (7) Persistent cyanosis. (8) Paralysis, especially of lower motor neurone type.

Collect: (1) Stomach wash (entire quantity). (2) 10 ml. blood. (3) Urine, 30 ml. 10 mg. of sodium fluoride for 10 ml. of blood acts both as a preservative and as an anticoagulant.

Symptoms of Chronic Poisoning: (1) The symptoms are exaggerated after the administration of suspected food, fluid or medicine. (2) Malaise, cachexia, depression and gradual deterioration of general condition of the patient is seen. (3) Repeated attacks of diarrhoea, vomiting, etc., are seen. (4) When the patient is removed from his usual surroundings, the symptoms disappear. (5) Traces of poison may be found in the urine, stool or vomit.

DIAGNOSIS IN THE DEAD

(I) Postmortem Appearances: (1) External: (1) The surface of the body and the clothes may show stains or marks of vomit, faeces or the poison itself. The colour changes in the corroded skin and mucous membrane are: (1) Sulphuric acid and hydrochloric acid: grey, becoming black from blood. (2) Nitric acid: brown. (3) Hydrofluoric acid: reddish-brown. (4) Carbolic acid: greyish-white. (5) Oxalic acid: grey, blackened by blood. (6) Cresols: brown, leathery. (7) Caustic alkalis: greyish-white. (8) Mercuric chloride: bluish-white. (9) Zinc chloride: whitish. (10) Chromic acid and potassium chromate: orange, leathery. **(2) Colour of postmortem staining:** The skin may be yellow or dark-brown in phosphorus and acute copper poisoning, cherry-red in poisoning by carbon monoxide, chocolate-coloured or brown in cases of death from poisoning by nitrites, aniline, nitrobenzene, acetanilide, bromates, chlorates, pyrogallol, MIC, due to the formation of methaemoglobin. **(3) Smell about the mouth and nose:** Substances which may be recognised by their odour are: (a) Garlic-like: phosphorus, arsine gas, arsenic (breath and perspiration), thallium, selenium, dimethylsufoxide, tellurium, parathion, malathion. (b) Sweet or fruity: ethanol, chloroform, nitrites. (c) Acrid: paraldehyde, chloral hydrate. (d) Rotten eggs: hydrogen sulphide, mercaptans, disulfiram. (e) Fishy or musty: zinc phosphide. Other substances are: cyanides, phenol, opium, ether, camphor, etc. **(4) The natural orifices,** e.g., mouth, nostrils, rectum and vagina may show the presence of poisonous material or the signs of its having been used. **(5) Injection marks** should be looked for with care. **(6) Skin** should be examined for lesions, e.g., hyperkeratosis and pigmentation may be found in chronic arsenical poisoning. Jaundice may occur in poisoning from senecio, phosphorus and in susceptible persons by potassium chlorate. **(7) Any evidence of marks of violence, such as bruises, or wounds of any nature** may suggest some form of death other than poison.

Internal: (1) Smell: On opening the body, note any peculiar smell. The skull should be opened first to detect unusual odours in the brain tissues. This is useful in cyanide, alcohol, phenol, cresol, ether, chloroform and camphor poisoning.

(2) Mouth and throat: Examine the tongue, and mouth for any evidence of inflammation, erosion or staining.

(3) Oesophagus: Corrosive alkalis produce marked softening and desquamation of the mucous membrane. In acute cantharidin poisoning, the mucous membrane is often swollen and engorged and may show patches of ulceration. Perforation of oesophagus may occur in poisoning from paraquat and fluorides.

(4) Upper respiratory tract: Examine the larynx, trachea and bronchi for evidence of volatile irritants or inhaled poisonous matter. **Oedema of glottis and congestion and desquamation of the mucous membrane of the trachea and bronchi may be seen in corrosive acid or alkali poisoning when it enters the respiratory tract.**

(5) Stomach: Toxic substances may be held in high concentrations in the rugae and crypts of the mucosa, or even in the blood in the actual stomach wall. The **pathway of acids and alkalis in food-filled stomach starts along the lesser curvature of the stomach and leads to the pylorus, which explains the location of greatest damage in food-filled stomach** (Figs 23-1 and 23-2). Stomach without food tend to have significant injury in the lower half to two-thirds and may have sparing of the fundus (Fig. 23-3).

(a) Hyperaemia: Hyperaemia of the mucous membrane **caused by an irritant poison is usually patchy and of a deep crimson colour.** The ridges are more involved. The mucous membrane is often covered with a sticky secretion and shows small haemorrhagic foci.

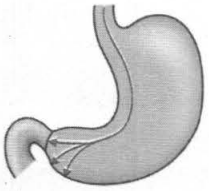


Fig. (23-1). Magenstrasse is the term applied to the pathway acidic agents follow.

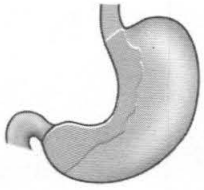


Fig. (23-2). Shaded region indicates the area of greatest injury caused by acid ingestion in a food-filled stomach.

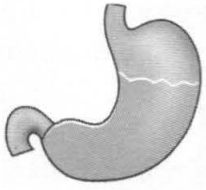


Fig. (23-3). Shaded region indicates the area of greatest injury caused by acid ingestion in an empty stomach.

(b) Softening: Corrosives and irritants produce immediate contraction of the muscularis, due to which the superficial epithelium is damaged, while the depths of the glands are protected by compression of their necks by the spasm. Excess of mucus is secreted by the glands due to the neighbouring irritation. If life is prolonged, the poison passes deeper and deeper. **Spasm of the pylorus holds a poison at this point which is the site most often involved. Softening of mucous membrane of stomach, is usually caused by corrosive poisons, chiefly alkaline corrosives.** It is also seen in mouth, throat and oesophagus. In disease, it is limited to stomach and is usually found at its cardiac end.

(c) Ulcers: Ulceration due to corrosive or irritant poisons appears as erosion with thin, friable margins. The surrounding mucosa is softened due to inflammation, and there is diffuse hyperaemia. An ulcer from disease is usually seen on the lesser curvature and the margins are well-defined, thickened and indurated.

(d) Perforation: Perforation is occasionally observed when the strong mineral acids have been taken, **especially sulphuric acid; it is much less common with the other acids. The stomach is blackened and extensively destroyed, the opening is irregular, the edges sloughing, and the adjacent tissues easily torn.** The acid escapes into the abdomen and causes peritonitis. Ammonia can cause perforation of stomach. Perforation by irritant poisons is rare. In chronic gastric ulcer, it is oval or rounded, and has a punched-out appearance, and may show chronic adhesion to neighbouring organs.

The contents of the Stomach: The stomach is opened along its greater curvature in a clean porcelain dish. The wall is examined for fragments of poisons adhered to it, such as powdered poisons, fragments of capsules, starch from tablets, fragments of leaves or fruits, cantharides, etc. The contents are observed regarding the volume, colour, etc., including food. The presence of seeds, leaves, capsules and foreign bodies, such as nails, pins, glass, etc., noted.

(6) The Duodenum and Intestines: A strongly acid reaction is of greater significance here than in the stomach contents. **Sodium hydroxide can rarely cause perforation of duodenum. The only characteristic change which occurs in the intestine**

is seen in mercury poisoning. This change which usually involves the ascending and transverse colons is a colitis, which may resemble the enteritis of acute bacillary dysentery.

(7) **Liver:** Substances, such as arsphenamine, chloroform, trinitrotoluene, carbon tetrachloride and senecio may produce liver necrosis. Arsenic, carbon tetrachloride, amanita phalloides, yellow phosphorus, iodine and rarely ferrous sulphate produce a fatty liver. Jaundice may be produced by phosphorus, senecio and potassium chlorate, due to acute hemolytic anaemia.

(8) **Kidneys:** Parenchymatous degenerative changes are commonly found in irritant metal poisoning and in cantharidin poisoning. Extensive necrosis of proximal convoluted tubules may be found in deaths from poisoning by mercuric chloride, phenol, lysol and carbon tetrachloride.

(9) **Heart:** Subendocardial haemorrhages in the left ventricle occur in most cases of acute arsenic poisoning.

(10) **The bladder** and in females the **vagina** and **uterus** should be particularly examined, for poison is occasionally introduced into the body by these routes.

The **lethal dose** is the dose that kills (Table 23-2). The usual lethal dose of a poison is 10 times the therapeutic dose.

Young's rule: The dose of a drug for a child is obtained by multiplying the adult dose by the age in years and dividing the result by the sum of the child's age plus 12.

$$\frac{\text{Adult dose} \times \text{child age}}{12 + \text{child age}}$$

(II) Chemical Analysis: Poison found in liver, kidney and blood is proof of absorption. Poison found in urine, unless added with evil intention, is a proof of absorption and excretion. The urine contains the drug or poison in many cases. It is suitable for single direct spot tests. In delayed deaths, the toxic agent may be found in the urine, when none is found in the viscera.

Putrefaction and Toxicologic Analysis: In postmortem decomposition, many poisons present in the tissues undergo chemical changes and cannot be detected by chemical analysis. Putrefaction of normal tissues may produce substances which give chemical reaction similar to those obtained from toxic compounds. Most volatile compounds are lost due to putrefaction, but **ethyl alcohol and cyanide may be produced from normal tissues.** Neurin, muscarin, mydalein, etc., are produced due to putrefaction, the toxicity of which is equal to the well-known alkaloids. In an embalmed body, it is very difficult to detect and identify most volatile poisons. CO, cyanides, organophosphates, endrin, datura, strychnine, yellow oleander, nicotine, fluorides and heavy metals resist putrefaction.

FAILURE TO DETECT POISON: In some cases, no trace of poison is found on analysis, although from other circumstances it is almost or quite certain that poison was the cause of illness or death. The **possible explanations of negative findings are:** (1) The poison may have been eliminated by vomiting and diarrhoea, e.g., in irritant poisons. (2) The whole of the poison has disappeared from the lungs by evaporation or oxidation. (3) The poison after absorption may be detoxified, conjugated and eliminated from the system. (4) Some vegetable alkaloidal poisons cannot be definitely detected by chemical analysis. (5) Some drugs are rapidly metabolised, making extraction difficult. (6) Some organic poisons especially alkaloids and glucosides may by oxidation during life or due to faulty preservation or a long interval, or from decomposition of the body may deteriorate and cannot be detected chemically. (7) Treatment given may alter the poisonous substance. (8) Biological toxins and venoms which may be protein in nature cannot be separated from body tissues. (9) Many drugs may be present in very small amount and these may require considerable amount of viscera for their identification. (10) The wrong or insufficient material may have been sent for analysis.

(III) Experiments on Animals: Cat and dog are affected by poisons almost in the same way as man. They may be fed with the suspected food, or with the poison after it is separated from the viscera and the symptoms noted.

(IV) Moral and Circumstantial Evidence: Such evidence may consist of motive, the evidence of witness about the recent purchase of the poison, his behaviour before and after the commission of the offence, and the recovery of poison from the possession of the accused.

DRUG AUTOMATISM: According to this hypothesis, **the patient develops a state of toxic delirium or amnesia after ingesting one or several doses of a drug (usually depressant drugs, alcohol**

or a combination of these), and in the delirious or automatism state, takes additional doses of the drug without realising it. It is difficult to prove or disprove this hypothesis.

DUTIES OF MEDICAL PRACTITIONER IN A CASE OF SUSPECTED POISONING: The duties are: (1) **Medical:** Care and treatment of the patient. (2) **Legal:** Assist the police to determine the manner of death. (1) Note preliminary particulars of the patient, i.e. name, age, sex, address, date and time, identification marks, etc. (2) In case of suspected homicidal poisoning, the doctor must confirm his suspicion before expressing an opinion. For this he must: (a) Collect vomit, urine and faeces and submit for analysis. (b) Observe and record the symptoms in relation to food; any change in the colour, taste or smell of the food or drink; other persons affected at the same time; the condition of the patient; explanation offered by the patient for the symptoms and statements of other persons present which appear to be relevant. (c) Consult in confidence senior doctor and keep him informed about the case. (d) Remove the patient to hospital. If the patient refuses to be moved, the doctor should appoint nurses of his confidence, who should themselves administer medicines and food, and maintain detailed records of the condition of the patient and of the treatment. No one should be allowed to stay with the patient alone. (e) If a particular person is suspected, attempts should be made to outwit him by changes of diet and the alteration of meal times. Such person should be allowed to visit the patient only in the presence of a nurse or a doctor. (f) Keep detailed records of the number of his visits, the symptoms and signs observed, and treatment given from time to time. Once the suspicion of homicide is confirmed, he should request the removal of the patient to hospital. (3) Any suspected articles of food, excreta, and stomach wash samples should be preserved. Full or empty bottles, capsules, paper packets, or liquids lying about should be collected and preserved. Any recent stains on bed clothes, furniture, etc., should be preserved. (4) **If a private practitioner is convinced that the patient is suffering from homicidal poisoning, he is bound under** (S. 39, Cr. P.C.) **to inform the police officer or Magistrate. (5) If he is sure that the patient is suffering from suicidal poisoning,** and if the condition of the patient is not serious, **he is not bound to inform the police.** (6) If the practitioner is summoned by the investigating police officer, he is bound to give all information regarding the case that has come to his notice. If he conceals the information or gives false information, he is liable to be prosecuted. (7) **A Government Medical Officer has to report to police all cases of suspected poisoning, whether accidental, suicidal or homicidal treated in the hospital.** (8) If the condition of the patient is serious, he must arrange to record the dying declaration. (9) If the patient dies, he should not issue a death certificate, but he should inform the police. In case of food poisoning, Public Health authorities must be notified.

TREATMENT OF POISONING

(I) Immediate resuscitative measures in comatose patients should be adopted to stabilise respiration, circulation and to correct CNS depression (ABCD of resuscitation). (A) **Airway:** Opening up and cleaning up the airway (oral cavity, nostrils) of secretions, vomit or any other foreign body by means of endotracheal intubation may be necessary. (B) **Breathing:** If the arterial blood gas cannot be maintained inspite of establishing an effective airway, then graduated supplemental oxygen therapy either by a ventimask or through endotracheal tube should be administered. (C) **Circulation:** I.V. fluid administration may be life sustaining line. (D) **Depression of CNS** should be corrected. Most of the poisoning cases, whether they are conscious or unconscious recover with supportive care alone.

An unconscious patient should be turned to lie on one side to stop the tongue blocking the throat and to allow fluid to come out of the mouth (recovery position).

(II) REMOVAL OF UNABSORBED POISON FROM THE BODY. (1) Inhaled Poisons: If the poison is inhaled as a gas, the patient must be removed into fresh air, artificial respiration and oxygen (6 to 8 litres per minute) should be given. The air-passages should be kept free from mucus by postural drainage or by aspiration. Nikethamide 2 ml. i.v. should be given if necessary. Give aminophylline 250 to 500 mg. if there is severe bronchospasm, and diuretics if there is pulmonary oedema.

(2) Injected Poisons: If the poison has been injected subcutaneously from a bite or an injection, a **tight ligature should be applied immediately above the wound,** which must be loosened every 10 minutes for one minute to prevent gangrene. The wound should be excised, the poison sucked out and the poison neutralised by suitable chemical substance. Local vasoconstriction can be produced by injection of adrenaline.

(3) Contact Poisons: Patient's contaminated clothes, contact lenses and jewellery should be removed immediately. If poison is applied to the skin or wound or is inserted into the vagina, rectum, or urinary bladder, **it should be removed by washing with water or should be neutralised by specific chemical.** Eyes should be irrigated with normal saline for at least 15 minutes.

(4) Ingested Poisons: **Gastric lavage is useful any time within three hours after ingestion of a poison. It is done using a stomach tube (Ewalds or Boa's tube), or ordinary soft, non-collapsible** rubber tube of one cm. diameter and one-and-half metres length, with a funnel attached at one end and a mark about 50 cm. from the other end, which should be rounded with lateral openings. At about the midpart of the tube there is a suction bulb, used to pump out the stomach contents. A wooden mouth gag has a hole at its mid-part to allow the passage of the tube through it. One end of the gag is pointed so that it can be forcefully inserted by the side of the mouth in non-cooperative patients (Fig. 23-4). **Dentures must be removed and a mouth gag is placed in right position in between the teeth of two jaws, so** that the teeth do not bite the tube in patients whose mouth cannot be kept open while passing the tube. Care should be taken in unconscious persons who are likely to regurgitate, and then aspirate stomach contents into respiratory tract and die from asphyxia. **Patient should be lying on his left side or prone with head hanging over the edge of the bed, and face down supported by an assistant,** so that the mouth is at a lower level than larynx. The end is lubricated with olive or sweet oil, or glycerine and is passed into the stomach by depressing the tongue with two fingers or tongue depressor and slowly **passing it downwards through the pharynx and oesophagus into the stomach till the 50 cm. mark is reached.**

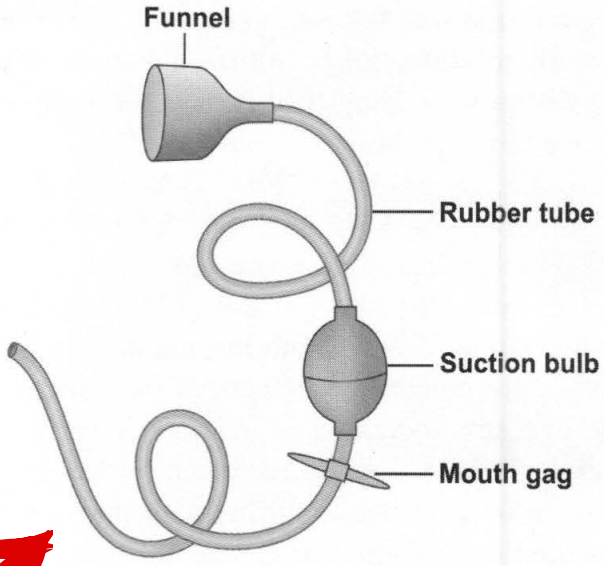


Fig. (23-4). Stomach pump.

If there are no marks on the tube, the tube should be passed for a distance equal to that measured between the bridge of the nose and the tip of the xiphoid process. Absence of coughing and of breath sounds in the funnel will confirm that the tube has not entered the trachea. Whenever in doubt, test by keeping the free end of the tube just below a water surface. Air from the stomach is usually expelled completely in two to three expirations, whereas air from the lungs causes bubbling at each expiration. **About 250 ml of warm water (35°C) should be passed through the funnel held high up above the patient's head. When funnel is empty, allow few minutes for fluid to act in the stomach, compress the tube below it between the finger and thumb, and lower it below the level of the stomach, and its contents will be emptied by syphon action on releasing the pressure on the rubber tubing. If stomach pump is used, applying suction on the bulb will siphon the stomach contents. Preserve this for chemical analysis.** Gastric lavage may be done with water; 1:5,000 potassium permanganate; 5% sodium bicarbonate; 4% tannic acid; one per cent sodium thiosulphate; one per cent sodium or potassium iodide; one to three per cent calcium lactate; saturated lime water or starch solution. Next, use about half litre of suitable solution and repeat till clear and odourless fluid comes out. This indicates that there is no further interaction between the antidote and poison. At this stage, the stomach is not completely emptied, but a small quantity of the

fluid containing the antidote or activated charcoal suspension (one gm/kg body weight) or/and a cathartic is left behind in the stomach, so that it may neutralise whatever small quantity of the poison is left behind. **Ryle's tube or a number 10 to 12 French catheter can be used for infants and children; about 25 cm. is necessary to reach the stomach.** After a recent heavy meal, the bulky contents are first removed by emetics. **Stomach wash is better than emesis because of the discomfort caused to the patient in vomiting.**

In poisoning with salicylates, phenothiazines, tricyclic antidepressants, lavage can be done up to 12 to 18 hours after ingestion of the poison.

Contraindications: (1) The **only absolute contraindication is corrosive poisoning (except carbolic acid)** due to the danger of perforation. **In the following conditions stomach wash can be done by taking proper precautions.** (2) **Convulsant poisons**, as it may lead to convulsions. Lavage should be done after controlling the convulsions. (3) **Comatose patients**, because of the risk of aspiration of fluid into the air-passages. The airway should be sealed by cuffed intubation and lavage done. (4) **Volatile poisons** which may be inhaled. (5) **Upper alimentary disease**, e.g. oesophageal varices. (6) In patients with **marked hypothermia, and haemorrhagic diathesis.**

Complications: (1) Laryngeal spasm. (2) Aspiration pneumonitis. (3) Perforation of stomach. (4) Sinus bradycardia.

EMETICS: Emetics should be used only if there is difficulty in obtaining or using a stomach tube. Due to the danger of inhaling gastric contents, vomiting should only be induced, when a conscious patient is lying on his side with the head dependent. **Ipecac powder one to two g. or 30 ml. of ipecac syrup for adults**, 15 ml (1 to 2 years), 10 ml (9 to 12 months), 5 mL (6 to 9 months) **followed by several glasses of water produces vomiting in 90 to 95% of patients within 20 to 30 minutes. The dose is repeated if vomiting does not occur in half an hour.** This is the only and best method of producing vomiting. Stimulation of the back of throat with finger, leaf, twig or a wooden tongue depressor is usually ineffective and may be dangerous. **Household emetics**, i.e. mustard powder and common salt are not effective and can lead to complications. Ingestion of excessive amount of salt water may cause fatal hypernatraemia. Apomorphine, copper sulphate, tartar emetic and zinc sulphate are obsolete.

Contraindications: Same as for stomach wash, and (1) Severe heart and lung diseases. (2) Advanced pregnancy. → Preterm delivery

(III) ADMINISTRATION OF ANTIDOTES: Antidotes are substances which counteract or neutralise the effects of poisons.

(1) Mechanical or Physical Antidotes: They **neutralise poisons by mechanical action or prevent their absorption.** (1) **Activated charcoal** is a fine, black, odourless and tasteless powder. It can be used by mixing with water to form soup-like slurry (8 ml of water per gram of charcoal). Superactivated charcoal has double the adsorbing surface area. **It acts mechanically by adsorbing and retaining within its pores organic and also to a less degree, mineral poisons and thus delays the absorption from the stomach.** Barbiturates, atropine, opioids, benzodiazepines, phenothiazines, amphetamines, antidepressants, antihistamines, antiepileptics, quinine, strychnine, digitalis and chloroquine are well adsorbed. Paracetamol, salicylates, kerosene and phenol are moderately adsorbed. It is not useful in poisoning with corrosives, heavy metals, cyanide, hydrocarbons and alcohol. **The initial dose is 60 to 100 g. in adults and 15 to 30 g. in children. Repeat doses of 50 g. every four hours can be given in poisoning by aspirin, phenobarbitol, theophylline and carbamazepine up to 2 days.** (2) **Demulcents are substances which form a protective coating on the gastric mucous membrane and thus do not permit the poisons to cause any damage, e.g. milk, starch, egg-white, mineral oil, milk of magnesia, aluminium hydroxide gel, etc.** Fats and oils should not be used for oil-soluble poisons, such as kerosene, phosphorus, organophosphorus compounds, DDT, phenol, turpentine, aniline, acetone, carbontetrachloride, etc. (3) **Bulky food** acts as a mechanical antidote to glass powder by imprisoning its particles within its meshes.

Multidose activated charcoal: It facilitates the passage of substances from plasma into the intestinal lumen (by creating a concentration gradient between the blood and bowel fluid), **where the concentration of toxin has been significantly lowered by intraluminal**

charcoal adsorption, and significantly decreases the half-life of several drugs. Initial loading dose is 1 to 2 g/kg. Repeat doses of 0.5 to 1 g/kg are given at 4 to 6 hours intervals. It can also be administered by continuous infusion of 0.25 to 0.5 g/kg/hour through a nasogastric tube.

CHARCOAL HAEMOPERFUSION: This is useful even with highly protein-bound substances that have a large volume of distribution and are lipid-soluble. They include barbiturates, salicylates, paraquat, phenytoin, theophylline, chloral hydrate, digitalis, glutethimide, methaqualone, methotrexate, pentobarbital, carbamazepine, theophylline and paracetamol. **Blood is circulated extracorporeally from an arterial source through a filter filled with adsorptive materials,** i.e. charcoal coated with various polymers (acrylic hydrogel is commonly used), or resins and then back to the patient's venous side. The circuit is heparinised and primed with saline.

(2) Chemical Antidotes: They counteract the action of poison by forming harmless or insoluble compounds or by oxidising poison when brought into contact with them. (1) Dialysed iron is used to neutralise arsenic. (2) Albumen precipitates mercuric chloride. (3) Common salt decomposes silver nitrate by direct chemical action, forming the insoluble silver chloride. (4) Copper sulphate is used to precipitate phosphorus. (5) Alkalis neutralise acids. It is safer to give a little weak solution of magnesia or ammonia. Bicarbonates should not be given because of the possible risk of rupturing the stomach due to liberated CO_2. (6) Acids neutralise alkalis. Only those substances which are by themselves harmless should be given, e.g. vinegar, lemon juice, canned fruit juice. Neutralisation of acids with alkali and vice versa should be avoided because exothermic reaction of neutralisation can cause additional injury. (7) Potassium permanganate has oxidising properties. 1:5000 solution is used in poisoning for opium and its derivatives, cyanides, barbituric acid and its derivatives, atropine and its derivatives, strychnine, phosphorus, hydrocyanic acid, and cyanides. When it reacts with the poison in the stomach, it loses its pink colour. The wash must be continued till the solution coming out of the stomach is of the same pink colour as the solution put in. (8) A solution of tincture iodine or Lugol's iodine 15 drops to half a glass of warm water precipitates most alkaloids, lead, mercury, silver, quinine and strychnine. (9) Tannic acid 4% or tannin in the form of a strong tea or one teaspoonful of tannic acid in water tends to precipitate apomorphine, cinchona, strychnine, nicotine, cocaine, aconite, pilocarpine, lead, silver, aluminium, cobalt, copper, mercury, nickel and zinc.

Universal antidote: So called **universal antidote** consisting of activated charcoal or burnt toast 2 parts; magnesium oxide one part; and tannic acid or strong tea one part is not recommended.

(3) Physiological or Pharmacological Antidotes: They act on the tissues of the body and produce symptoms exactly opposite to those caused by the poisons. They are used after some of the poison is absorbed into the circulation. Their use is somewhat limited and not without danger. **These agents act on the principle of antagonism,** utilising when possible the power which different drugs possess of interfering with another's action upon the enzymes, tissue cells or opposing nerve systems. Most of the known antidotes are only partial in their action. **Atropine and physostigmine are two real physiological antidotes,** as both of them affect nerve endings and produce opposite effects on the heart rate, state of the pupils, and glandular secretory activity. Other examples are: **barbiturates and picrotoxin or amphetamine; strychnine and barbiturates; cyanides and amyl nitrite.**

Chelating Agents: Chelating agents (metal complexing agents) are used in the treatment of poisoning by heavy metals. They have a greater affinity for the metals as compared to the endogenous enzymes. **The complex of the agent and metal is more water soluble than the metal itself, resulting in higher renal excretion of the complex.**

(A) B.A.L. (British anti-Lewisite; dimercaprol; 2, 3, mercaptopropanol): It is used as a physiological antidote in arsenic, lead, bismuth, mercury, copper, gold and other heavy metals. **Many heavy metals have great affinity for sulphydryl (–SH) radicles and combine with them in tissues, and deprive the body of the use of respiratory enzymes of tissue cells. Dimercaprol has two unsaturated sulphydryl groups which combine with the metal, and thus prevent union of arsenic with the –SH group of**

the respiratory enzyme system. The compound formed by the heavy metal and dimercaprol is relatively stable which is carried into the tissue fluids, particularly plasma and is excreted in the urine. In severe poisoning, a dose of **3 to 4 mg/kg. is given.** Three ml. of 10% B.A.L. and 20% benzyl benzoate in arachis oil is **injected deep intramuscularly fourth hourly for the first two days, and then twice daily for 10 days or till recovery.** It should not be used when liver is damaged.

(B) E.D.T.A. (ethylenediamine tetra-acetic acid; calcium disodium versenate; edathamil; edetic acid): It is a chelating agent and is effective in lead, mercury, copper, cobalt, cadmium, iron and nickel poisoning. The usual dose is **25 to 35 mg/kg. body weight in 250 to 500 ml. of 5% glucose or normal saline i.v. over a one to two hour period twice daily for five days,** and may be repeated after two to three days. It forms chelates with lead which are water-soluble, non-toxic, non-ionised, non-metabolised and excreted intact in the urine. It is superior to B.A.L. for the treatment of poisoning by arsenic and mercury. It is the treatment of choice in lead poisoning.

(C) PENICILLAMINE (cuprimine): It is a hydrolysis product of penicillin. It has a stable SH group. It is given in a dose of **30 mg/kg. body weight up to a total of two g. per day in 4 divided doses orally for about 7 days.** One to three g. can be given in slow normal saline drip daily for 2 to 4 days. It is the chelating agent of maximum efficiency for copper, lead and mercury.

(D) DMSA (Meso-2,3-dimercapto succinic acid; **succimer**): It is used in lead, mercury and arsenic poisoning. **It is superior to EDTA in the treatment of lead poisoning,** as it does not lead to redistribution of lead to the brain. It is less toxic to the kidneys. It can be given in patients with 6-PGD deficiency, where use of BAL is contraindicated. It is given in a dose of **10 mg/kg orally every 8 hours for 5 days,** followed by the same dose every 12 hours for 14 days. A combination of succimer and EDTA is said to be more effective.

(E) DMPS (2,3-dimercaptopropane 1-sulfonate) is effective in the treatment of mercury, lead and arsenic poisoning. It is given in a dose of **5 mg/kg. i.v. in 6 divided doses, followed by 100 mg. orally twice a day for 24 days.**

DMSA and DMPS possess the same dithiol (sulphydryl) chelating grouping as dimercaprol and the molecules are more hydrophilic. They have a better therapeutic index.

(IV) ELIMINATION OF POISON BY EXCRETION: INDICATIONS: (1) Severe poisoning. (2) Progressive deterioration in spite of full supportive care. (3) When there is high risk of serious morbidity or mortality. (4) When normal route of excretion of the toxic compound is impaired. (5) When the poison produces delayed but serious toxic effects. (6) When the patient is having cardiovascular, respiratory or other diseases that increase the hazards.

(1) RENAL EXCRETION: It may be improved by giving large amounts of fluid, tea or lemonade orally. Forced diuresis may cause pulmonary or cerebral oedema, and is not recommended. Urinary acidification is not recommended.

(2) PURGING: 30 g. sodium sulphate with large amounts of water, hastens the elimination of poison in the stool. Magnesium sulphate should be avoided, since sufficient may be absorbed to produce CNS depression in cases of renal failure. To remove unabsorbed material from the intestinal tract, poorly absorbable material, such as liquid petroleum which is a solvent for fat-soluble agents is effective. Sorbitol 50 ml of 70% solution is a better purgative, but in young children it may cause fluid and electrolyte imbalance.

(3) WHOLE BOWEL IRRIGATION: Whole bowel irrigation involves the use of a polyethylene glycol with electrolyte lavage solution which is a nonabsorbable, osmotically active compound. This is administered usually by nasogastric tube (0.5 litre/hour to children less than 5 years of age and 2 litre/hour to adults) continuously until the rectal effluent is clear. It takes about 2 to 4 hours. It is useful in patients who have ingested large quantities of substances that are difficult to remove, e.g., iron and lithium overdose, sustained release preparations, cocaine, heroin, etc.

(4) DIAPHORETICS: In most cases application of heat (blankets, hot water bottles), and administration of hot beverages (hot tea, hot milk, hot lemonade) will cause increased perspiration. Profuse perspiration will be produced by five mg. of pilocarpine nitrate S.C. and a less marked effect may be produced by cutaneous irritation and cutaneous vasodilation produced by alcohol, salicylates and antipyretics.

(5) FORCED ALKALINE DIURESIS, achieving a urinary pH of 7.5 to 9 promotes excretion of drugs that are weak acids, such as salicylates, phenobarbital, chlorpropamide, methotrexate, etc. A solution of sodium bicarbonate 50 to 100 mEq. added to one litre of 0.45% saline may be administered at 250 to

500 ml/hr for the first 1 to 2 hours. Alkaline solution and diuretics should be administered to maintain a urinary output of 2 to 3 ml/kg/hr.

(6) PERITONEAL DIALYSIS: Alcohols, long-acting barbiturates, chloral hydrate, lithium, salicylates, bromides, inorganic mercury, quinidine, theophylline, and sodium chlorate are effectively removed by peritoneal dialysis. For adults, the exchange is usually 2 litre; for children under 5 years 200 ml. It is only 10 to 25% as effective as haemodialysis. Exchange transfusion especially in children is useful in barbiturate, CO and salicylate poisoning.

(7) HAEMODIALYSIS: It is very useful for removing ethanol, methanol, ethylene glycol, chloral hydrate, lithium, trivalent arsenic, acetaminophen, bromides, phenobarbital, salicylates, fluoride, sodium chlorate, digitalis, methaqualone, boric acid and thiocyanates.

(V) SYMPTOMATIC TREATMENT: It refers to the adoption of general measures to support the life of the patient and to lessen suffering. The symptoms should be treated on general lines.

(VI) Adequate follow-up is necessary to treat the complications if any. In suicidal cases, psychiatric treatment is necessary.

CHAPTER

24 Agricultural Poisons

ORGANOPHOSPHORUS POISONS

Compounds: They are derived from phosphoric acid and form two series of compounds.

(A) Alkyl phosphates: (1) HETP. (2) TEPP (tetron). (3) OMPA. (4) Dimefox. (5) Isopestox. (6) Malathion (kill bug; bugsoline). (7) Sulfotep. (8) Demeton (9) Tichlorfon. **(B) Aryl phosphates:** (1) Parathion (nitrostigmine), (follidol, kill phos; ekato). (2) Paraoxon. (3) Methyl parathion (metacide). (4) Chlorothion. (5) Diazinon (tik 20; diazion). They are available as dusts, granules and liquids.

Absorption: It is absorbed by inhalation, through the skin, mucous membranes and the gastrointestinal tract. When sprayed in air, absorption in the plants occurs through leaves and stems.

Action: Organophosphorus compounds are powerful inhibitors of carboxylic esterase enzymes, including acetylcholinesterase. They bind firmly to the esterase enzyme, inactivating it by phosphorylation at the myoneural junctions and synapses of the ganglions and inhibit AchE in all parts of the body, due to which acetylcholine accumulates at the parasympathetic, sympathetic and somatic sites, and transfer of nerve impulses across synapses at the autonomic ganglia at the nerve-muscle junction is prevented. Phosphorylated acetylcholinesterase loses an alkyl group, due to which the enzyme cannot spontaneously hydrolyse and becomes permanently inactivated. **They are called cholinesterase inhibitors.** This produces a syndrome of overactivity due to unhydrolysed acetylcholine. **Symptoms appear in both the sympathetic and parasympathetic nervous system.** Mild poisoning usually occurs when cholinesterase activity is 20 to 50% of normal; moderate poisoning occurs when activity is 10 to 20% of normal; severe poisoning when activity is less than 10% normal. Symptoms are similar to those resulting from overdosage of acetylcholine, pilocarpine, physostigmine or muscarine. They have three distinct toxic actions. (1) **A muscarine-like effect.** **(2) Nicotine-like stimulation, followed by paralysis** of preganglionic and somatic motor nerves. **(3) CNS stimulation followed by depression.**

Signs and Symptoms: Onset of systemic symptoms is most rapid following inhalation and least rapid following absorption from the skin. **With massive ingestion or inhalation, symptoms may begin within five minutes, or may be delayed for half to one hour and are at a maximum in 2 to 8 hours.** The respiratory or gastrointestinal symptoms are more marked depending on the route of entry.

(I) Muscarinic manifestations: These symptoms can be easily remembered by the acronym SLUDGE: Salivation, lachrymation, urination, defecation, gastrointestinal distress, and emesis. **Bronchial tree:** Bronchoconstriction, increased bronchial secretions, dyspnoea, cyanosis, pulmonary oedema. **G.I.:** Anorexia, nausea, vomiting, cramps, diarrhoea, faecal incontinence, tenesmus. **Sweat glands:** Increased sweating. **Salivary glands:** Increased salivation. **Lacrimal glands:** Increased lacrimation. **C.V.S.:** Bradycardia, hypotension. **Pupils:** Miosis, occasionally unequal or dilated. **Ciliary body:** Blurred vision. **Bladder:** Urinary incontinence.

(II) Nicotinic manifestations: (1) **Striated muscle:** Muscular fasciculations, cramps, weakness, areflexia, muscle paralyses; (2) **Sympathetic ganglia:** Hypertension, tachycardia, pallor, mydriasis.

(III) CNS manifestations: Restlessness, emotional lability, headache, tremors, drowsiness, confusion, slurred speech, ataxia, generalised weakness, coma, convulsions, depression of respiratory and cardiovascular centres.

Mild poisoning: Signs and symptoms are: nausea, malaise, fatigue, minimal muscle weakness, cramping without diarrhoea.

Moderate poisoning: SLUDGE and/or tremors, weakness, fasciculations, confusion, lethargy, anxiety.

Severe poisoning: SLUDGE and respiratory insufficiency, weakness, fasciculations, coma, paralysis, seizures, autonomic dysfunction.

Intermediate Syndrome: In some cases after one to four days, muscle weakness and paralysis characterised by motor cranial nerve palsies, weakness of neck, flexor and proximal limb muscles, and acute respiratory paresis are seen due to prolonged cholinesterase inhibition and muscle necrosis. It does not respond to oximes or atropine.

Chronic Poisoning: It usually occurs as an occupational hazard in agriculturists. The main features are: muscle cramps, weakness, gait disorders, paraesthesias, drowsiness, confusion, irritability and anxiety.

Porphyrinaemia, resulting in **chromolachryorrhoea** (shedding of red tears) due to accumulation of porphyrin in the lachrymal glands is seen rarely.

Delayed sequelae: Delayed peripheral neuropathy can occur one to 5 weeks after exposure to certain compounds, such as parathion, malathion, trichlorfon, etc. It begins with paraesthesias and pain or cramps in the calves followed by ataxia, weakness and toe drop. It rapidly progresses to a flaccid paresis which can ascend similar to Guillain-Barre syndrome. Reflexes are diminished. The disease may progress for 2 to 3 months, and muscle wasting occurs.

Fatal Dose:

TEPP: 50 mg i.m. or 100 mg. orally.
OMPA: 80 mg. i.m. or 175 mg. orally.
Parathion: 80 mg. i.m. or 175 mg. orally.
HETP: 60 mg. i.m. or 350 mg. orally.
Malathion and diazinon one g. orally.

Fatal Period: Usually within 24 hours in untreated cases. In non-fatal cases the acute effects last for 6 to 36 hours, which disappear in 2 to 3 days, but may sometimes persist for two weeks. Complete recovery occurs in ten days in patients treated early.

Cause of Death: Death is caused by paralysis of respiratory muscles, respiratory arrest due to failure of respiratory centre, or intense bronchoconstriction. Late death, as long as 15 days after acute ingestion may be caused by ventricular arrhythmias.

Diagnosis: (1) It depends on the signs and symptoms. (2) It may be confirmed by giving two mg of atropine. In a normal person this causes marked atropinisation, but in a case of poisoning by organophosphorus compound, symptoms are relieved without atropinizing. (3) Red cell and plasma cholinesterase levels of less than 50%.

Treatment: (1) The patient is removed from the source of exposure, the contaminated clothing removed and the exposed areas are washed with soap and water, followed by ethanol and water, or some alkaline solution. If eye is contaminated irrigate the conjunctival recesses, cornea, bulbar conjunctiva, internal and external palpebral surfaces. (2) The airway should be kept patent. Tracheostomy may be required. (3) When the poison is ingested, the stomach should be washed with 1:5,000 potassium permanganate solution. (4) Activated charcoal one g/kg. ~~Ipecac~~ should not be used. A cathartic, e.g. sorbitol or magnesium citrate can be given once unless diarrhea has occurred. (5) Avoid ~~physostigmine~~, ~~endorphonium chloride~~ and ~~succinylcholine~~. (6) **Atropine sulphate arrests the muscarine effects of postganglionic parasympathetic (peripheral) activity (muscarinic receptor antagonist), and arrests CNS effects.** It has no effect on nicotinic actions. It is ineffective on respiratory center in the presence of severe asphyxia and also if B.P. falls, Heart rate exceeding 140 beats/min should be avoided. **2 to 4 mg. is given i.v.** (paediatric dose 0.05 mg/kg.) as a test dose. If there is no effect this dose may be doubled every 5 to 10 minutes until muscarinic symptoms are relieved. **Atropine should be continued until the tracheobronchial tree is cleared of the secretions and most secretions are dried, but not pupillary status.** The average patient requires 40 mg per day, but as much as 1,000 mg/day has been used. It can be given in continuous infusion. Tachycardia is not a contraindication. Once signs of adequate atropinisation occur, the dose should be adjusted to maintain this effect for at least 24 hours. When cyanosis is present, maximal oxygenation should be achieved before atropine is given for avoiding increased risk of ventricular tachyarrhythmias associated with hypoxia. (7) **Specific cholinesterase reactivators like diacetyl monoxime (DAM), or 2-pyridine aldoxime methiodide (pralidoxime iodide, 2-PAM), or pralidoxime chloride (Protopan, 2-PAM chloride) or pyridine aldoxime methane sulphonate (P_2S) act by competing for the phosphate moiety of**

the organophosphorus compound and release it from the cholinesterase enzyme. DAM crosses the blood-brain barrier and can regenerate some of the CNS cholinesterase. Its action is marked at nicotinic sites, often improving muscle strength within 10 to 40 minutes. It also decreases muscarinic and CNS symptoms. **The adult dose is one to 2 g. i.v., either as a 5% solution given over 5 min., or in 150 ml. of saline and infused over half-an-hour.** This can be repeated in one hour if muscle weakness and fasciculations are not relieved. This dose should be repeated at 6 to 12 hours intervals for 24 to 48 hours to ensure distribution to all affected sites. **Maximum dose should not exceed 12 g. in a 24 hour period.** Alternatively, 2.5% solution can be given as continuous infusion of half g/hour. If signs and symptoms of cholinergic excess persist, continued dose may be necessary until symptoms subside. **Pralidoxime and atropine work synergistically, and should be used together. Oximes reactivate inhibited cholinesterase, remove the block at neuromuscular junction, prevent formation of phosphorylated enzyme, and directly detoxify organophosphates.** They do not cross blood-brain barrier. They should be started as early as possible, although they may be beneficial when given after 24 to 36 hours of exposure. They are given until the patient is clinically well and not requiring atropine. **Pralidoxime decreases the amount of atropine required and also potentiates the action of atropine.** Oximes are ineffective or minimally effective for: dimefox, dimethoate, ciodrin, methyl diazinon, methyl phenocapton, phorate, schradan and wepsyn. (8) **Obidoxime chloride is more potent, but its toxicity is slightly greater. Dose: 250 mg i.v. or i.m. It can be given if oximes are not available. (9) HI-6 and H10-7 appear to have activity against all known organophosphates.** (10) Convulsions can be controlled with i.v. diazepam 10 to 20 mg at a rate of 0.5 ml (5 mg per minute), repeated if necessary after 30 to 60 minutes. This may be followed by i.v. infusion to a maximum of 3 mg/kg of body weight over 24 hours. If convulsions persist, use i.v. phenobarbital 10 to 20 mg/kg body wt. or i.v. phenytoin 18 mg/kg. For status epilepticus, general anaesthesia may be used. Diazepam decreases anxiety and counteracts some aspects of CNS toxicity which are not affected by atropine. (11) Pulmonary oedema and bronchospasm should be treated with oxygen, intubation, atropine and positive pressure ventilation. (12) Antibiotics to prevent pulmonary infections.

Prophylaxis: The precautions to be taken are: (1) Protective clothing including overall of white cotton, a white cloth hood to cover the head and neck, rubber apron, gloves and boots, eyeshields and respirator. (2) The face and the hands should be thoroughly washed after spraying with soap and water, or he should take a bath on the farm itself. (3) Not more than two hours spraying a day should be done by a worker, and he should not work for more than six successive days on spraying. A person suffering from cold, bronchitis, etc., should not be engaged in spraying operations. (4) The workers should be properly instructed and their work supervised. (5) The workers should not smoke, chew, or drink in the spraying area. (6) Spraying machines, tanks, containers, hoses, etc., should be thoroughly washed at the end of the work and before repairs are carried out.

Postmortem Appearances: **Signs of asphyxia are found.** The face is congested and there is cyanosis of the lips, fingers and toes. Blood-stained froth is seen at the mouth and nose. The stomach contents may smell of kerosene. The mucosa of the stomach is congested with submucous petechial haemorrhages. Respiratory passages are congested and contain frothy haemorrhagic exudate. The lungs show gross congestion, excessive oedema and subpleural petechiae. Heart is sometimes soft and flabby. The internal organs are congested. The brain is congested and oedematous, and meninges are congested; petechial haemorrhages are present. The cholinesterase in erythrocytes and at myoneural junctions is below normal.

Poisoning: Certain groups of people are at risk. (1) Those who are engaged in the manufacture and packing of the compounds. (2) Those who use these compounds as sprays or dusts in the open as insecticides. (3) Children of users. (4) Research workers. (5) Suicide is very common. (6) Homicide is rare.

CARBAMATES

They are marketed in the form of dust or solutions, such as aldicarb (Temik), aminocarb (Matacil), aprocarb (Baygon), carbaryl (Sevin), carbofuran

(Furaxdan). **Absorption occurs through all routes. They will spontaneously hydrolyse from the cholinesterase enzymatic site within 48 hours, and as such CNS toxicity is limited.** Symptoms begin in 15 minutes to 2 hours. They are partially metabolised in the liver and directly excreted in the urine, faeces and milk. All other **clinical manifestations are similar to organophosphates.**

Treatment: **Atropine is the specific antidote.** Pralidoxime may diminish the severity of symptoms and help prevent some morbidity. It improves respiratory functions and patient's well-being.

ORGANOCHLORINES

Classification: The organochlorine (chlorinated hydrocarbons) can be divided into four categories: (1) DDT and analogues: DDT, methoxychlor. (2) Benzene hexachloride: gamma hexachlorobenzene (Lindane). (3) Cyclodienes and related compounds: aldrin, chlordane, chlordecone, dieldrin, endosulfan, endrin, heptachlor, isobenzan, mirex. (4) Toxaphene and related compounds. They are anticholinergic and inhibit carboxylic esterase enzymes by carbamylation. **All of these pesticides are absorbed through skin, orally and via inhalation.** DDT is the least well absorbed. These agents are highly lipid soluble. Endrin is rapidly metabolised and eliminated and does not persist in body tissues.

ENDRIN

Action: **They are anticholinergic and inhibit carboxylic esterase enzymes by carbamylation.**

Endrin belongs to the group of cyclodiene insecticides and is a polycyclic, polychlorinated hydrocarbon. It is a stereo-isomer of dieldrin. Its taste is bitter. It is also called **plant penicillin,** because of its broad spectrum of activity against various insect pests. It is sold in the market under the trade names of Endrin-we-16, Endox-DB 50, Endtox Ec-20, Endrex, Tafdrin, etc. These products contain about 20 to 50% of endrin mixed with petroleum hydrocarbon, such as aromax, which smell like kerosene.

Symptoms: They begin within one to six hours. They are salivation, nausea, vomiting, abdominal pain, rarely diarrhoea, hoarseness of voice, coughing, froth at the mouth and nose, dyspnoea, headache, giddiness, restlessness, hyperirritability, dilated pupils, incoordination, ataxia, mental confusion, tremors, tonic and clonic convulsions, coma and death due to respiratory failure. In non-fatal cases most of the persons feel well after 24 hours.

Fatal Dose: Five to six g.; DDT, gammexane; lindane: 15 to 30 g.

Fatal Period: 24 hours.

Treatment: (1) Gastric lavage, or the stomach evacuated by emetics, and cathartics. (2) Give animal charcoal. (3) **There is no specific antidote.** (4) Maintain and assure adequate airway, breathing and circulation. (5) If the mental state is altered give dextrose, naloxone and thiamine. (6) Clothing should be removed and skin washed with soap and water. (7) Control convulsions with diazepam followed by phenobarbital. (8) Calcium gluconate is useful. (9) Cholestyramine is a non-absorbable bile acid binding anion exchange resin, which increases the faecal excretion of organochlorines. It is given 16 g. per day in divided doses.

Postmortem Appearances: The mouth and **stomach contents smell of kerosene. Signs of asphyxia** are present.

Poisoning: Suicide is very common in spite of its unpleasant taste and painful death, because it is easily available. Homicide is rare.

FLUORIDES: **Sodium fluoride is used in rat poison and cockroach powders. Sodium silicofluoride, fluoroacetamide and fluoroacetate are used as rodenticides. Fluoride compounds react with acid in the stomach and form hydrofluoric acid which is corrosive. After absorption, fluoride ions bind calcium ions and to some extent potassium and magnesium ions and cause hypocalcaemia, hypokalaemia and hypomagnesaemia. Fluoride ions also inactivate proteolytic and glycolytic enzymes and act as general protoplasmic poison. Ingestion causes burning pain in the mouth and epigastrium, thirst, salivation, vomiting, diarrhoea, haematemesis and haematuria. Death may occur within minutes from respiratory and cardiac failure.**

Treatment: (1) Calcium orally and intravenously. (2) Stomach wash with lime water or milk.

Fatal Dose: 5 mg/kg. body weight.

ZINC PHOSPHIDE: It is a steel-grey, crystalline powder with a garlicky odour. It reacts with the acid in the stomach and liberates phosphine. The symptoms are: vomiting, diarrhoea, cyanosis, fever, respiratory distress and death.

It is used as rat poison, which causes death due to asphyxia.

Fatal Dose: 5 g.

Fatal Period: One day.

Treatment: (1) Stomach wash with potassium permanganate. (2) Purgatives.

Postmortem Appearances: Garlicky odour in stomach contents. Blood is cherry-red. Lungs are congested and oedematous and liver shows fatty degeneration and necrosis.

PARAQUAT

It is a dipyridylium compound used as herbicide and weed-killer. It is sprayed on weeds before planting crops. It is inactivated when in contact with soil. It is produced commercially as a brownish concentrated liquid of the dichloride salt in 10 to 30% strength, under the trade name, "Gramoxone" and as brown granules called "Weedol" at about 5% strength. Deaths occur due to ingestion. Deaths by inhalation while spraying are very rare.

ACTION: Paraquat undergoes a NADPH (nicotinamide adenine dinucleotide phosphate) dependent reduction to form the free radical, which reacts with molecular oxygen to reform the cation and produce a superoxide free radicals which disrupt cellular function, structure and cell death. Concentrate solution corrodes GI mucosa.

ABSORPTION AND EXCRETION: Absorption is mainly through GI tract and occurs through inhalation; skin or eye contact is minimal. Five to ten percent of the dose is absorbed, and the rest is excreted in the faeces. It is distributed to all the organs. More than 90% of the absorbed paraquat is excreted unchanged in the urine within the first 24 hours.

Fatal Dose: 3 to 5 g.

Fatal Period: 2 to 7 days.

SIGNS AND SYMPTOMS: LOCAL: Irritation and inflammation of skin, nails, cornea, conjunctivae and nasal mucosa. G.I.T.: Oropharyngeal ulceration and corrosion, nausea, vomiting, haematemesis, diarrhoea, painful mucosal ulceration, dysphagia, aphonia, perforation of oesophagus, mediastinitis and pneumothorax. RENAL: Oliguria or non-oliguric renal failure due to acute tubular necrosis. Lungs: Cough, haemoptysis, dyspnoea due to pulmonary oedema, haemorrhage or fibrosis. Pancreas: Pancreatitis. Liver: Centrilobular hepatic necrosis and cholestasis. C.V.S.: Hypovolaemia, shock, arrhythmias. C.N.S.: Late coma, convulsions, cerebral oedema. Adrenal: Insufficiency due to necrosis. BONE MARROW: Polymorphonuclear leucocytosis early: anaemia late.

CAUSE OF DEATH: Death occurs from multiorgan failure or corrosive effects in the G.I. tract. Death from oesophageal perforation and mediastinitis can occur within two to three days of ingestion. In ingestions of less than 3 g. death occurs from five days to several weeks.

TREATMENT: (1) Gastric lavage. (2) One litre of 15 to 30% aqueous suspension of Fuller's earth or 7% bentonite is given to adsorb paraquat, followed by 200 ml. of 20% mannitol. (3) If the above adsorbents are not available, activated charcoal can be given. (4) Haemodialysis and haemoperfusion is useful if done within 12 hours of ingestion. (5) Remove all clothing and wash the patient thoroughly with soap and water. (6) Analgesics should be given for pain.

POSTMORTEM APPEARANCES: There may be ulceration around the lips and mouth. The stomach may show erosions. The kidneys may show cortical pallor and diffuse tubular damage. Progressive lung damage may cause death within two weeks. Diffuse pulmonary oedema and haemorrhages occur. Within a few days repair begins. If patient continues to survive, the alveoli begin to fibrose, with reticulin and collagen being laid down to form a rigid, stiff lung. The lungs may be mistaken for a diffuse pneumonia. There may be a fibrinous pleurisy and sometimes slight bloody pleural effusion.

PYRETHRINS AND PYRETHROIDS

They are extracted from Chrysanthemum plant. Pyrethroids are synthetic analogues. They are used as insect repellents, insecticides and pesticides. Toxicity is very low due to their rapid metabolism. They are available as sprays, dusts, powders, mats and coils.

FATAL DOSE: 1 g/kg. body weight.

ACTION: They prolong the inactivation of the sodium channel by binding to it in the open state.

SIGNS AND SYMPTOMS: Skin contact causes dermatitis and blistering. Paraesthesias, nausea, vomiting, vertigo, fasciculations, hyperthermia, altered mental state, convulsions, pulmonary oedema and coma. Inhalation causes rhinorrhoea, sore throat, wheezing, and dyspnoea.

TREATMENT: (1) Stomach wash with activated charcoal. (2) Oils and fats should be avoided. (3) Atropine and oximes are contraindicated. (4) Skin should be washed with soap and water.

ALUMINIUM PHOSPHIDE

Aluminium phosphide (ALP) is a solid fumigant pesticide, insecticide and rodenticide It is widely used as grain preservative. In India it is available as white tablets of Celphos, Alphos, Quickphos, Phostoxin, Phosphotex, etc. each weighing three gm. and has the capacity to liberate one gm. of phosphine (PH_3).

Action: On coming in contact with moisture ALP liberates phosphine. The chemical reaction is accelerated by the presence of hydrochloric acid in the stomach. ALP has garlicky odour.

Absorption and Excretion: Phosphine is rapidly absorbed from the GI tract by simple diffusion and causes damage to the internal organs. It is also rapidly absorbed from the lungs after inhalation. Phosphine is oxidised slowly to oxyacids and excreted in the urine as hypophosphite and in unchanged form through the lungs.

Action: Phosphine inhibits respiratory chain enzymes. **It acts by inhibiting the electron transport resulting from preferential inhibition of cytochrome oxidase.**

Fatal Dose: 1 to 3 g; one to three tablets.

Fatal Period: One hour to four days. Majority die within 24 hours.

Signs and Symptoms: **(1) Inhalation:** Mild inhalation exposure produces irritation of mucous membrane and acute respiratory distress. Other symptoms are dizziness, easy fatigue, tightness in the chest, nausea, vomiting, diarrhoea and headache. Moderate toxicity produces ataxia, numbness, paraesthesia, tremors, diplopia, jaundice, muscular weakness, incoordination and paralysis. Severe toxicity produces adult respiratory distress syndrome, cardiac arrhythmias, congestive heart failure, pulmonary oedema, convulsions and coma.

(2) Ingestion: Mild intoxication produces nausea, vomiting, headache and abdominal pain, and the patients usually recover. Moderate and severe poisoning produces: **G.I.T.:** Nausea, vomiting, diarrhaea, retrosternal pain. **C.V.S.:** Hypotension, shock, arrhythmias, myocarditis, pericarditis, acute congestive heart failure. **R.S.:** Cough, dyspnoea, cyanosis, pulmonary oedema, respiratory failure. **Hepatic:** Jaundice, hepatitis, hepatomegaly. **Renal:** Renal failure. **C.N.S.:** Headache, dizziness, altered mental state, restlessness, convulsions, acute hypoxic encephalopathy, coma.

Treatment: (1) Gastric lavage with potassium permanganate is done and repeated two or three times to oxidise the poison. (2) Give activated charcoal 100 g. orally to adsorb phosphine. (3) Antacids reduce symptoms pertaining to the stomach and reduce absorption of phosphine. (4) Liquid paraffin is given for excretion of ALP and phosphine from the gut. (5) **There is no specific antidote.** (6) Magnesium sulphate reduces organ toxicity, corrects hypomagnesaemia and arrhythmias. The usual dose is one g. repeated for the next two hours, and then one to 1.5 g. every 6 hours for 5 to 7 days in the form of continuous i.v. infusion. (7) Low dose dopamine infusion, 4 to 6 micrograms/kg/min. is useful. (8) I.V. hydrocortisone, 400 mg. every 4 to 6 hours is highly effective. It reduces the dose of dopamine. (9) Adequate hydration and renal perfusion must be maintained with i.v. fluids. (10) Hypoxia is treated with oxygen. (11) Shock should be treated with fluids and hydrocortisone. (12) Metabolic acidosis should be corrected with i.v. sodium bicarbonate. (13) Peritoneal or haemodialysis is useful.

Postmortem Appearances: Garlic-like odour is present at the mouth and nostrils and in the gastric contents. Blood-stained froth is found at the mouth and nostrils. Mucous membrane of lower part of oesophagus, stomach and duodenum are congested. Decreasing congestion of the G.I. tract is seen in the small intestine. The lungs, liver, spleen, kidneys and brain are congested.

CHAPTER

25 Corrosive Poisons

Action: A **corrosive poison** fixes, destroys and erodes the surface with which it comes into contact. They act by extracting water from the tissues, and coagulate cellular proteins, and convert haemoglobin into haematin.

MINERAL ACIDS

Action: **They produce coagulation necrosis. Hydrofluoric acid causes liquefaction necrosis. They have no remote action. They act as irritants when slightly diluted, but as stimulants when well diluted.**

Acids produce more damage to the stomach than the oesophagus. No trace of acid may be found if the victim survives for two days or more.

Stains on Skin: **Nitric acid produces a yellowish stain, sulphuric acid and caustic alkalis reddish-brown or dirty-brown stains, hydrochloric acid and carbolic acid whitish or greyish-white stains on the skin and mucous membranes.**

SULPHURIC ACID (oil of vitriol)

Physical characters: Pure sulphuric acid (H_2SO_4) is a heavy, odourless, colourless, non-fuming, hygroscopic, oily liquid and has tendency to carbonize organic substances. Commercial sulphuric acid is usually brown or dark in colours. It causes superficial burns after only one second of contact, and full thickness burns after 30 seconds.

Signs and Symptoms: **The lips are usually swollen and excoriated, and brown or black streaks may be found extending from the angles of the mouth to the sides of the chin,** and sometimes to the front of the neck due to flow of the acid. **There is corrosion of mucous membranes of mouth, throat and oesophagus,** immediate burning pain and dysphagia. Epigastric pain soon spreads all over the abdomen and thorax. **Pharyngeal pain is the most common presenting symptom.** Eructation, nausea and vomiting occur. The vomit is brown or black, mucoid, strongly acid and may contain shreds of the charred wall of the stomach. Thirst is intense, but any attempt to drink causes vomiting. Spitting and dribbling of the acid from the mouth cause erosion of the skin over the chin, angles of the mouth and hands. **Teeth are chalky-white. Tongue becomes swollen, sodden and black.** The abdomen becomes distended and very tender. Constipation is severe and there is tenesmus. The voice becomes hoarse. The eyes are sunken and the pupils usually dilated. The mind remains clear till death. If person recovers, late oesophageal, gastric and pyloric strictures and stenoses may develop.

Fatal Dose: 10 to 15 ml.

Fatal Period: 12 to 24 hours.

Cause of Death: (1) Circulatory collapse. (2) Spasm or oedema of glottis. (3) Collapse due to perforation of stomach. (4) Toxaemia. (5) Delayed death may occur due to hypostatic pneumonia, secondary infection, renal failure or starvation due to stricture of oesophagus.

Complications: Perforation of stomach, stricture oesophagus, secondary infection, renal failure.

Treatment: (1) Avoid gastric lavage or emetics. (2) **The acid should be immediately diluted and neutralised *in situ* by giving one-fourth litre of water or milk or milk of magnesia or lime water or soap suds or aluminum hydroxide gel if the patient is seen within 30 minutes of ingestion.** Alkaline carbonates and bicarbonates, which liberate carbon dioxide, should not be used, as they cause gastric distention and sometimes rupture. (3) Give a demulcent: olive oil, milk, egg-whites, starch water, mineral oil, melted butter. (4) Prednisolone 60 mg/day may be given in divided doses to prevent oesophageal stricture and for shock. Later, 3 to 4 cm. diameter mercury-filled bougie should be passed daily if stricture develops. (5) Correct circulatory shock. (6) Tracheostomy, if there is oedema of glottis. (7) Give nothing by mouth. Nutrient substances are given by intravenous route for about a week. Then try liquids, soft food, and finally a regular diet. (8) Skin burns are washed with large amounts of water, and a paste of magnesium oxide or sodium bicarbonate is applied. (9) Eye burns are irrigated with water or

sodium bicarbonate solution for 10 to 15 minutes. A suspended i.v. bag that administers low pressure irrigation is ideal. (10) Symptomatic.

Postmortem Appearances: External: They depend on the quantity and strength of the acid used, and the time that the patient survives after taking the acid. Corrosion of mucous membranes of lips, mouth and throat and of the skin over the chin, angles of the mouth and hands is seen. The necrotic areas are at first greyish-white, but soon become brown or black and leathery. The clothing should be examined for burns and stains.

Internal: Internal changes are limited to the upper digestive tract, and the respiratory system. The upper digestive tract is inflamed and swollen by oedema, and severe interstitial haemorrhage, even when corrosion is absent. Perforation of the oesophagus is rare. **The greater part of stomach may be converted into a soft, spongy, black mass which disintegrates when touched. Sometimes, only the pyloric region is involved** because fluid pathway from oesophagus occurs usually along lesser curvature to the pylorus. Initial exposure of the pylorus to acid causes severe spasm, which promotes injury at this site. **The mucosal ridges are more damaged than the intervening furrows. In the damaged area, the mucosa or even the whole thickness of the stomach wall has a brown or black colour. Perforation may occur with the escape of gastric contents into the peritoneal cavity,** and if the patient lives for few hours, chemical peritonitis and corrosion of organs is seen. Perforation of diaphragm may occur. The duodenum may show similar but less intense changes, and the small intestine may show signs of irritation. In many cases, little or no free acid can be found in the viscera, because the acid is converted to substances normally present in the body. **Corrosion or severe inflammation of the larynx and trachea may be present.**

Poisoning: (1) Accidental poisoning results due to mistaking it for glycerine or castor oil. (2) Most cases are suicidal. (3) It is not used for homicide. (4) It is taken internally or injected into vagina as abortifacient.

VITRIOLAGE (vitriol throwing): Throwing of sulphuric acid on another individual is known as vitriolage (Plate 20-119). Jealous or disgruntled persons may throw a corrosive to disfigure and harm their enemies. Blindness may occur if the eyes are involved. Death may result from shock or toxaemia, if extensive area is involved. **They are penetrating burns and the acid devitalises the tissues and as such are relatively painless, but predispose to infection.** Repair is slow and the scar tissue causes contracture. Sometimes, nitric acid, carbolic acid, corrosive alkali or juice of marking nut or calotropis is used to disfigure the face.

Treatment: The affected part is washed with plenty of water and soap or sodium or potassium carbonate. Later, a thick paste of magnesium oxide or carbonate is applied. The eyes are washed with water and irrigated with a dilute sodium bicarbonate solution. Later, a few drops of olive oil or castor oil are put into the eyes.

NITRIC ACID (HNO_3)

Nitric acid (aqua fortis, red spirit of nitre) is a clear, colourless, fuming, heavy liquid, and has a peculiar and choking odour. **In concentrated form, it combines with organic matter and produces a yellow discolouration of tissue due to the production of picric acid (xanthoproteic reaction).**

Signs and symptoms are those of poisoning by sulphuric acid. There is more eructation and greater abdominal distension, owing to gas formation. It causes yellow discolouration of the tissues, including the crowns of the teeth and yellow stains on the clothing. Inhalation of fumes causes lachrymation, photophobia, irritation of air-passages and lungs producing sneezing, coughing, dyspnoea and asphyxia. Colour of urine is brown.

Fatal Dose: 10 to 15 ml.

Fatal Period: 12 to 24 hours.

Treatment: Same as for sulphuric acid.

Postmortem Appearances: They are those of sulphuric acid, but the tissues are stained yellow. There is corrosion of mucous membrane of the oesophagus and stomach, which may appear brown or brown-black due to acid haematin, but not yellow. The stomach is soft, friable and ulcerated. Perforation of the stomach is not common but extensive areas of the mucosa of the stomach or oesophagus are sometimes detached. In death from inhalation of fumes, the larynx, trachea, and bronchial tubes are congested and lungs are oedematous.

HYDROCHLORIC ACID (HCL)

Hydrochloric acid (muriatic acid) is a pungent, colourless, fuming liquid.

Signs and Symptoms: It is less violent in its action than sulphuric acid. It does not usually corrode or seriously damage the skin, but it readily destroys mucous membranes. The mucous membrane is at first grey or grey-white, and later becomes brown or black, due to the production of acid haematin. Inhalation of fumes causes intense irritation of throat and lungs with symptoms of suffocation, coughing, dyspnoea and cyanosis.

Fatal Dose: 15 to 20 ml.

Fatal Period: 18 to 36 hours.

Treatment: Same as for sulphuric acid.

Postmortem Appearances: They are those produced by sulphuric acid although corrosion is less severe. The stomach contains brownish fluid; the folds of the whole stomach mucosa are brownish. Perforation of the stomach is rare. Acute inflammation and oedema of respiratory passages and lung tissue are common.

OXALIC ACID

Physical characters and uses: Oxalic acid (acid of sugar, $C_2H_2O_4$) occurs in the form of colourless, transparent, prismatic crystals, which resemble in appearance the crystals of magnesium sulphate and zinc sulphate. **In the form of oxalate, it exists as a natural constituent of many plants, e.g., spinach, rhubarb, cabbage, etc.** About 20 mg. is excreted in urine daily. It is used as a bleach to remove stains, or to clean brass and copper articles or leather, in calico-printing and for removing iron moulds from linen. It is also used for removing writing and signature illegally.

Action: Local: Crystals of the acid and concentrated solution of oxalates are corrosive poisons. They rarely damage the skin, but readily corrode the mucous membrane of the digestive tract. They do not lose their poisonous properties when diluted. **Dilute solutions act as mild local irritants, but cause serious systemic effects when absorbed.**

Systemic: (a) Shock: Large doses cause rapid death from shock. **(b) Hypocalcaemia:** Those who survive for a few hours develop hypocalcaemia because it readily combines with the calcium ion in the body tissues and causes withdrawal of calcium from the tissues. **(c) Renal damage:** Oxalates produce tubular nephrosis or necrosis and cause death from uraemia in 2 to 14 days.

Fatal Dose: 15 to 20 g.

Fatal Period: One to two hours.

Signs and Symptoms: (a) Fulminating Poisoning: Large concentrated dose of 15 g. or more produce immediate symptoms and death within minutes. There is a burning, sour, bitter taste in the mouth with a sense of constriction around the throat and burning pain from the mouth to the stomach. Pain is very severe, begins in the epigastrium, but soon radiates all over the abdomen; there may be tenderness. Nausea and eructation are immediately followed by vomiting which may be persistent. Vomit usually contains altered blood and mucus and has a "coffee-ground" appearance. Thirst may be present. Death usually occurs before bowels are affected, but if life is prolonged diarrhoea will occur.

(b) Acute Poisoning: When the patient survives for a few hours after a large dose, the symptoms are those of hypocalcaemia, and less by digestive upset. **There is muscle irritability and tenderness, tetany or usually convulsions. There may be numbness and tingling of the fingertips and legs.** Usually, signs of cardiovascular collapse appear. In some patients, stupor and coma occur.

(c) Delayed Poisoning: The symptoms are of uraemia. The urine may be scanty or suppressed and may contain traces of blood, albumin, and calcium oxalate crystals. There may be metabolic acidosis and ventricular fibrillation.

Treatment: (1) The stomach is washed out carefully using calcium lactate or gluconate, two teaspoonfuls in each lavage. (2) **The antidote for oxalate poisoning is any preparation of calcium which converts the poison into insoluble calcium oxalate,** e.g. lime water, calcium lactate, calcium gluconate, calcium chloride, a suspension of chalk in water or milk. One-and-half g. of chalk will neutralise about one gram of oxalic acid. (3) Calcium gluconate 10%, 10 ml. i.v. at frequent intervals. (4) Dialysis or exchange transfusion for renal failure. (5) **Parathyroid extract 100 units i.m. in severe cases.** (6) Demulcent drinks. (7) The bowels may be evacuated by an enema or by castor oil. (8) Symptomatic.

Postmortem Appearances: Burns of the face and skin are rare. **If the poison has been used in strong solution, the mucous membrane of the tongue, mouth, pharynx and oesophagus will be whitened as if bleached, and has a scalded appearance,** but is sometimes reddened by irritation. The inner surface of the oesophagus is corrugated. The outer coats of the stomach may be inflamed. The mucous membrane of the stomach is reddened or punctate from erosions or almost black. It may be softened in patches, but perforation is very rare. Many dark-brown or black streaks run along the length of the stomach over the mucous membrane, often with intercommunicating branches. The stomach contents are gelatinous and brownish due to acid haematin formation. Sometimes, the whole stomach will become corroded. The intestines usually escape, but upper part of the duodenum may be affected. The kidneys are swollen by oedema, congested and the tubules are filled with oxalate crystals. If the effects are only narcotic, there will be congestion of the lungs, liver, kidneys and brain, without any local appearances.

The Circumstances of Poisoning: (1) Accidental poisoning is due to its being mistaken for magnesium sulphate, or sodium bicarbonate. (2) Suicidal and homicidal poisoning is rare. (3) Rarely, it is used to procure abortion by vaginal injection.

CARBOLIC ACID (phenol)

Physical characters and uses: When pure, the acid consists of short, colourless, prismatic, needle-like crystals, which have a burning sweetish taste, which turn pink and liquefy when exposed to air. It has a characteristic "carbolic or phenolic" smell. It is slightly soluble in water, but is freely soluble in glycerine, ether, alcohol, and benzene. The commercial carbolic acid is a dark-brown liquid containing several impurities, chiefly cresol. It is largely used as an antiseptic in various preparations and as a disinfectant.

Important derivatives of phenol include cresol, creosote (coal tar), lysol, dettol, thymol, menthol and tannic acid.

Absorption: **It is absorbed from the alimentary tract, respiratory tract, rectum, vagina, serous cavities, wounds and through the skin.**

Elimination: **Phenol is converted into hydroquinone and pyrocatechol in the body before being excreted in the urine.** A trace is excreted by the lungs, salivary glands, skin and stomach. About 36 hours are required for complete excretion.

Fatal Dose: 10 to 15 g.

Fatal Period: 3 to 4 hours.

Signs and Symptoms: Poisoning by carbolic acid is known as **corbolism.**

Local: (1) Skin: It causes **burning and numbness due to damage to nerve endings.** It precipitates protein and coagulates the cell contents. It produces **a white opaque eschar which is painless** and falls off in a few days and leaves a brown stain. There may be necrosis and gangrene of the tissue which becomes green-white or brown-white; the dead tissue sloughs readily.

(2) Digestive Tract: Hot burning pain extends from the mouth to the stomach, which is followed by tingling and later anaesthesia. **Deglutition and speech become painful and difficult.** The lips, mouth and tongue are corroded, which soon become white and hardened. Nausea and vomiting are present in about 20% cases.

(3) Respiratory Tract: Pulmonary and laryngeal oedema develop due to irritation.

Systemic Effects: **Phenol is a depressant of the nervous system, especially the respiratory centre.** Headache, giddiness, tinnitus, muscular spasm and later collapse, unconsciousness and coma occur. The temperature is subnormal, the pupils are contracted, breathing is stertorous, pulse is rapid, feeble and irregular, face covered with cold sweat, and there is dusky **cyanosis, respiratory alkalosis and metabolic acidosis.** Liver may be damaged. In severe cases, methaemoglobinaemia is a characteristic feature. There is a strong odour of phenol in breath.

Urine: It is scanty and contains albumin and free haemoglobin; suppression may follow. **It may be colourless or slightly green at first, but turns green or even black on exposure to air.** In the body, phenol is partly oxidised to hydroquinone and pyrocatechol, which with unchanged phenol are excreted in the urine, partly free, and partly in unstable combination with sulphuric and glucoronic acids. **The further oxidation of hydroquinone and pyrocatechol in**

the urine is the cause of green colouration. This is known as carboluria.

Oochronosis: The hydroquinone and pyrocatechol may cause **pigmentation in the cornea and various cartilages, a condition called oochronosis.** Oochronosis is commonly associated with alkaptonuria, in which homogentic acid gets deposited in cartilages, ligaments and fibrous tissues.

Chronic poisoning is characterised by anorexia, weight loss, headache, vertigo, dark urine and pigmentation of skin.

Cause of Death: (1) Syncope. (2) Asphyxia due to (a) failure of respiration, (b) oedema of glottis, (c) complications, e.g. bronchopneumonia.

Treatment: (1) **An ~~emetic~~ often fails due to the anaesthetic effect.** (2) The stomach should be washed carefully with plenty of lukewarm water containing animal charcoal, olive oil, castor oil, magnesium or sodium sulphate, or saccharated lime with which phenol combines and forms harmless products. Soap solution or 10% glycerine may be used, and the washing continued until the washings are clear and odourless. This is followed by administration of olive oil or vegetable oil to remove surface phenol and to prevent deep penetration. (3) When lavage is completed, 30 g. of magnesium sulphate or a quantity of medicinal liquid paraffin should be left in the stomach. (4) Demulcents. (5) Activated charcoal in usual dose. (6) Haemodialysis if there is renal failure. (7) Methylene blue i.v., if there is severe methaemoglobinaemia. (8) Saline containing 7 g. of sodium bicarbonate per litre is given i.v. to combat circulatory depression, to dilute carbolic acid content of blood and to encourage excretion by producing diuresis. (9) If phenol falls on the body, contaminated clothing should be removed at once, the skin washed with undiluted polyethylene glycol. If this is not available, the area washed with soap and water for at least 15 minutes. Olive oil, or methylated spirit, or 10% solution of ethyl alcohol act as solvents which should be wiped out.

Postmortem Appearances: External: Corrosion of the skin, especially in tracks from the angles of the mouth on to chin, has a greyish or brown colour. Signs of splashing may be seen elsewhere. **The tongue is usually white and swollen**, and there is smell of phenol about the mouth. **The mucous membrane of the lips, mouth and throat is corrugated, sodden, whitened or ash-grey and partially detached with numerous small submucous haemorrhages.**

Internal: The mucosa of the oesophagus is tough, white or grey, corrugated and arranged in longitudinal folds. The stomach mucosal folds are swollen and covered by opaque, coagulated grey or brown mucous membrane. The intervening furrows are usually less damaged, dark-red and are not opaque. **The mucous membrane is thickened and looks leathery** (Plate 21-126). Often there is partial separation of necrotic mucosa, with severe congestion of underlying tissue. The stomach may contain a reddish fluid mixed with mucus and shreds of epithelium emitting an odour of phenol. The duodenum and upper part of the small intestine may show similar but milder changes. **The liver and spleen usually show a whitish, hardened patch where the stomach has been in contact with them due to the transudation of phenol.** The kidneys show haemorrhagic nephritis in cases of delayed death. The brain is congested and may be oedematous. The blood is dark and semifluid or only partially coagulated. If vomit or poison has been inhaled, coagulation necrosis of the mucosa and severe congestion of the submucosa of the air-passages may be seen. Laryngeal and pulmonary oedema also occur.

The Circumstances of Poisoning: (1) It is used for suicidal purposes. (2) A~~ccidenta~~l poisoning is rare. (3) It is rarely used for ~~homicide~~ because of its odour and taste. (4) It is sometimes injected into the vagina and uterus to produce abortion.

BORIC ACID: It is used as antiseptic and insecticide. 15 to 20 g. is fatal.

Symptoms: Generalised erythema resembling "boiled lobster" appearance with massive areas of desquamation indistinguishable from toxic epidermal necrolysis or staphylococcal scaled syndrome is seen.

Detergent or chemical suicide: Common household chemicals (bath sulphur with toilet bowl cleaner) are mixed to produce hydrogen sulphide gas in cars, closets or other enclosed spaces.

CAUSTIC ALKALIS

The chief poisons are ammonia, potassium hydroxide, sodium hydroxide, calcium hydroxide, ammonium carbonate, potassium carbonate and sodium carbonate. Most of these occur as white powders. Ammonia is a colourless gas with a very pungent, choking odour.

Ammonium hydroxide is a liquid containing about 30% ammonia.

ACTION: Strong alkali absorbs water from the tissues and precipitates protein. Alkalis produce liquefaction necrosis and saponification of fats. Ingestion of an alkali produces severe effects mainly on lining of oesophagus, while gastric involvement is less common. Therefore, stricture formation is much more common with alkalis than with acids.

SIGNS AND SYMPTOMS: In general, the lesions caused by caustic alkalis have about the same extent and distribution as those due to acid corrosives. There is an acid caustic taste, and a sensation of burning heat extending from the throat to the stomach. Alkalis produce a soft, oedematous, translucent, soap-like, swollen eschar, red-brown from the absorption of altered blood pigment. The sloughs are mucilaginous. Charring is not seen. The vomited matters are alkaline and do not effervesce on contact with the ground. It is at first thick and slimy, but later contains dark altered blood and shreds of mucosa. Purging is a frequent symptom, accompanied by severe pain and straining. Oesophageal stricture formation is common with alkalis than acids.

Contact with skin causes greyish, soapy, necrotic area. When strong alkali is ingested, abrasions, blisters and brownish discolouration are seen on the lips and the skin about the mouth. The mucosa of the digestive tract is swollen and a grey slough readily detached, lies over the inflamed tissues. Haemorrhage into the tissue is also seen.

Ammoniacal vapour, when inhaled, causes congestion and watering of the eyes, violent sneezing, coughing and choking. Sudden collapse and death may occur from suffocation due to inflammation and much swelling of the glottis or later from pneumonia.

FATAL DOSE: 5 to 30 g.; Ammonia: 5 to 10 ml.

FATAL PERIOD: Usually 24 hours.

TREATMENT: (1) Neutralise poison by giving vegetable acids, e.g., acetic, citric, or tartaric acid mixed with large quantity of water. (2) Demulcents. (3) In mild cases the stomach can be washed carefully. (4) In poisoning by ammonia vapour, oxygen inhalation should be given or the patient should be kept in an atmosphere made moist with steam. (5) Symptomatic.

POSTMORTEM APPEARANCES: The marks about the mouth become dark in colour and parchment like after death. When a strong alkali is ingested, lips, mouth and throat show corrosion. Inflammatory oedema with corrosion and sliminess of the tissues of the oesophagus and stomach are prominent features. Mucosa may be brownish due to formation of alkali haematin (Plate 21- 121). The duodenum and jejunum may show similar changes but of lesser intensity. In some cases, the alkali may be regurgitated and inhaled causing oedema of the glottis, pseudomembranous inflammation of the air-passages and a peribronchial pneumonia. Perforation of the oesophagus or stomach is rare, but may occur in ammonia poisoning.

LYE (NaOH) can produce transmural necrosis of oesophagus after only one second of contact. Oesophageal stricture is common with occasional perforation.

CIRCUMSTANCES OF POISONING: Poisoning by alkalis is rare. Homicidal poisoning is rare, but few suicidal cases are seen. Poisoning by ammonia is more common than other alkalis.

CHEMICAL BURNS: They are produced by acids and alkalis. Prolonged contact with cement and with hydrocarbons, such as gasoline can cause chemical burns. They are also produced by phosphorus and phenol.

CHAPTER

26 Metallic Poisons

ARSENIC

Metallic arsenic is not poisonous, as it is not absorbed from the alimentary canal.

Poisonous Compounds: **(1) Arsenious oxide or arsenic trioxide: (*sankhya or somalkhar*): It is commonly known as white arsenic or arsenic. It occurs as white, smooth, heavy, crystalline powder or as solid mass. It has no taste or smell and is sparingly soluble in water. It sublimes on heating. (2) Copper arsenite (Scheele's green), and copper acetoarsenite (Paris green or emerald green). (3) Arsenic acid. (4) Sodium and potassium arsenate. (5) Arsenic sulphide, orpiment and realgar. (6) Arsenic trichloride. (7) Arseniuretted hydrogen or arsine is a colourless gas with garlic-like non-irritating odour. (8) Organic compounds, e.g. cacodylates, atoxyl, acetarson, tryparsamide, salvarsan, mepharsen, etc.**

Action: **It combines with sulphydryl enzymes, and interferes with cell metabolism.** Locally it causes irritation of the mucous membranes and remotely depression of the nervous system.

Signs and Symptoms: **(1) The Fulminant Type:** Large dose (3 to 5 g) of arsenic when rapidly absorbed causes death in one to three hours from shock and peripheral vascular failure. In this type gastrointestinal symptoms are absent.

(2) The Gastroenteric Type: **This is the common form of acute poisoning, and resembles bacterial food poisoning.** Symptoms usually appear half to one hour after ingestion, but may be delayed many hours especially when taken with food. There is sweetish metallic taste. **G.I.T.:** Constriction in the throat and difficulty in swallowing, burning and colicky pain in oesophagus, stomach and bowel occur. **Intense thirst and severe vomiting which may be projectile are the constant symptoms.** Purging is usually accompanied by tenesmus, and pain and irritation about the anus. **The stools are expelled frequently and involuntarily** and are dark-coloured, stinking and bloody, but **later become colourless, odourless and watery resembling rice-water stools of cholera** (Table 26-1). **Hepatic:** Fatty infiltration. **Renal:** Oliguria, uraemia. Urine contains albumin, red cells and casts, pain during micturition. **C.V.S.:** Acute circulatory collapse with vasodilation, increased vascular permeability, ventricular tachycardia, ventricular fibrillation. **CNS:** Headache, vertigo, hyperthermia, tremors, convulsions, coma, general paralysis. **Skin:** Delayed loss of hair, skin eruptions.

(3) Narcotic Type: In this, the gastrointestinal symptoms are very slight. There is giddiness, formication and tenderness of the muscles, delirium, coma and death. Rarely, there is complete paralysis of the extremities.

Areseniuretted hydrogen, when inhaled acts as a direct poison to the haemoglobin, producing haemolysis, haemoglobinuria and renal failure. Death is almost instantaneous. Haemodialysis is effective.

Fatal Dose: 100 to 200 mg for arsenic trioxide.

Fatal Period: One to two days.

Treatment: (1) Emetics can be given. (2) The stomach should be emptied and then repeatedly washed with large amount of warm water and milk. The stomach should be washed out at intervals to remove iron compounds, and adherent arsenic. (3) Butter and greasy substances prevent absorption. (4) Alkalis should not be given as they increase the solubility of arsenic. (5) **Freshly precipitated hydrated ferric oxide orally in small doses converts toxic arsenic to nontoxic ferric oxide.** (6) **BAL** 400 to 800 mg. on first day, 200 to 400 mg. on second and third days in divided doses every 4 hours, and 100 to 200 mg. in two divided doses for 7 to 10 days. (7) **Calcium disodium versenate is superior to BAL in some respects.** (8) **Penicillamine** 100 mg./kg. daily in four divided doses for five days. This should be given if BAL or DMSA are not available. (9) **DMSA (succimer) 10 mg/kg orally every 8 hours for 5 days, followed by the same dose every 12 hours for 14 days. It is superior to BAL.** (10) DMPS. (11) Haemodialysis or exchange transfusion

Table (26-1). Difference between arsenic poisoning and cholera

Trait	Arsenic poisoning	Cholera
(1) Pain in throat:	Before vomiting.	After vomiting.
(2) Purging:	After vomiting.	Before vomiting.
(3) Stools:	Dark-coloured and bloody, later rice-watery.	Rice-watery, not bloody and passed in continuous involuntary jet.
(4) Tenesmus and anal irritation:	Present.	Absent.
(5) Vomited matter:	Contains mucus, bile and blood.	Watery without mucus, bile and blood.
(6) Voice:	Not affected.	Rough and whistling.
(7) Conjunctivae:	Inflamed.	Not inflamed.
(8) Analysis of excreta:	Arsenic present.	*Cholera vibrio* present.
(9) Circumstantial evidence:	Of arsenic poisoning may be present.	Other cases of cholera in locality.

if necessary. (12) Chelation therapy is ineffective in arsine poisoning.

Postmortem Appearances: External: The eyeballs are sunken and the skin is cyanosed. The body may be shrunken due to dehydration.

Internal: The lesions are mainly found in the stomach. The mucosa is swollen, oedematous and red, either generally or in patches, especially in the pyloric region. **There may be lines of redness running along the walls or curved lines of submucous haemorrhages. Usually groups of petechiae are seen scattered over the mucosa, and sometimes large submucosal and subperitoneal haemorrhages. The stomach mucosa resembles red velvet.** Small acute ulcerations or large erosions may be found, especially at the pyloric end. A mass of sticky mucus covers the mucosa in which particles of arsenic may be seen. Congestion is most marked along the crests of the rugae. The small intestine appears flaccid and contains large flakes of mucus with very little faecal matter. **The mucosa is pale violet and shows signs of inflammation with submucous haemorrhages along its whole length.** The caecum and rectum show slight inflammation. Sometimes, arsenic penetrates through the walls of the stomach and appears in liver, omentum and endocardium. **If putrefaction has taken place, yellow streaks will be found in the subperitoneal layer of the stomach and to less extent of the intestines, due to absorbed arsenic which has been converted into sulphide.** In fulminating type, the stomach and intestines may not show any signs of inflammation. The liver, spleen and kidneys are congested, enlarged and show cloudy swelling and occasionally fatty change. The lungs are congested with subpleural ecchymoses. Nephritis, particularly of glomerular type is frequent. Haemorrhages may be found in the abdominal organs, mesenteries, and occasionally in larynx, trachea and lungs. There may be oedema of brain with patchy necrosis or haemorrhagic encephalitis. The meninges are congested. **Subendocardial haemorrhages of the left ventricle are common in arsenic poisoning and may be found even when the stomach does not show signs of irritation.** X-ray may show presence of arsenic in GI tract.

Chronic Poisoning: It may be due to accidental ingestion of repeated small doses by those working with the metal or by taking food or drink in which there are traces of drug. It may be of homicidal nature due to repeated small doses, or may occur after recovery from one large dose. **CNS:** Polyneuritis, optic neuritis, anaesthesias, paraesthesias. **Skin: Pigmentation consists of a finely mottled brown change mostly on the temples, eyelids and neck (rain drop type of pigmentation)** which persists for many months. There may be a rash resembling fading measles rash (Plate 19-114). **In prolonged contact, hyperkeratosis of the palms and soles with irregular thickening of the nails and development of bands of opacity in the fingernails called**

Aldrich-Mees lines is seen. **Eyes:** Congestion, watering of the eyes, photophobia. **G.I. Tract:** Cirrhosis of the liver, nausea, vomiting, abdominal cramps, diarrhoea, salivation. **C.V.S. and kidneys:** Chronic nephritis, cardiac failure, dependent oedema. **Hepatic:** Hepatomegaly, jaundice. **Haematologic:** Bone marrow suppression, hypoplasia, anaemia, thrombocytopaenia, leucopaenia, basophilic stippling (rarely). **R.S.:** Cough, haemoptysis, dyspnoea. Arsenic is teratogenic, and can result in lung and skin cancer, leukaemia, etc.

Organic Arsenical Compounds: They are less toxic than inorganic compounds. Their chief toxic manifestations are immediate anaphylactic symptoms and later on skin reactions, agranulocytosis, hepatitis, jaundice and encephalitis.

Absorption: It is absorbed from the alimentary tract and from the skin when applied in a suitable vehicle and by inhalation.

Distribution: In the early stage, it is found in greatest quantity in the liver followed by kidneys and spleen. **Arsenic is excreted into the stomach and intestines after absorption even when given by routes other than mouth.** In cases in which life is prolonged it is found in the muscles for days, in the bones and in the keratin tissues, hair, and nails for years. Neutron activation analysis and atomic absorption spectroscopy help in estimating concentration of arsenic in hair, nails, bones, etc.

Elimination: It is eliminated mainly by the kidneys, but also in the faeces, bile, sweat, milk and other secretions. ~~Breast milk~~ does not contain significant amounts of arsenic. Arsenic is excreted into the epidermal tissues, and in cases of intermittent chronic poisoning there will be successive deposits of arsenic in the hair and nails.

Tolerance: Some people take arsenic daily as tonic or as an aphrodisiac and they acquire tolerance up to 0.3 g. or more in one dose. Such people are known as arsenophagists.

The Circumstances of Poisoning

(I) Homicide: Arsenic is the most popular homicidal poison because: (1) it is cheap, (2) easily obtained, (3) colourless, (4) no smell, (5) no taste, (6) small quantity is required to cause death, (7) can be easily administered with food or drink, (8) onset of symptoms is gradual, (9) symptoms simulate those of cholera. **The disadvantages are:** (1) it delays putrefaction, (2) can be detected in completely decomposed bodies, (3) can be found in bones, hair and nails for a long time, (4) can be detected in charred bones or ashes. (5) **Mass homicidal poisoning occurs when it is mixed with food or in a well.** (6) Sometimes, it is given mixed with tobacco cigars for homicide or to rob.

(II) Suicide is ~~rare~~.

(III) Accident: (1) Admixture with articles of food or from its improper medicinal use. (2) Chronic poisoning results from drinking well water containing arsenic. (3) It is sometimes ingested or applied locally in the form of a paste or ointment to abortion sticks to produce abortion.

It is fed to the animal mixed with cattle fodder.

Postmortem Imbibition of Arsenic: In exhumations, the possibility of imbibition of arsenic from the stomach into neighbouring viscera and also contamination from the surrounding earth should be remembered. Arsenic found in the soil is usually an insoluble salt. **Keratin tissues absorb arsenic by contamination from outside.** If arsenic is introduced into the stomach after death, the transudation occurs into the organs of the left side before those of the right and the signs of ~~inflammation~~ and ~~ulceration~~ are absent.

ANTIMONY

Metallic antimony is not poisonous. Antimony potassium tartrate (tartar emetic) and antimony trichloride are poisonous. The mode of action and symptoms of acute and chronic poisoning are similar to those produced by arsenic.

Fatal Dose: Tartar emetic 0.2 to 0.5 g; antimony trichloride 0.1 to 0.2 g.

Fatal Period: Usually within 24 hours.

Treatment: (1) Stomach wash. (2) Tannic acid 4 g. by mouth forms an insoluble antimony tannate. (3) B.A.L. (4) Symptomatic.

MERCURY (Quick silver)

Physical characters: It is liquid metal, bright silvery appearance and is volatile at room temperature. It forms two series of compounds: (1) mercuric, which are soluble and intensely poisonous, and (2) mercurous, which are much less soluble and therefore less active. Metallic mercury is not poisonous if swallowed, for it is not absorbed. If the mercury is breathed or swallowed as vapour or if applied to the skin or mucous membrane in finely divided state it is absorbed.

POISONOUS COMPOUNDS: (1) Mercuric chloride (corrosive sublimate) occurs as colourless masses of prismatic crystals or as a white crystalline powder. It has no smell, but a styptic, nauseous, metallic taste. (2) Mercuric oxide (brick-red crystalline powder). (3) Mercuric iodide (scarlet-red powder). (4) Mercuric cyanide (white prismatic crystals). (5) Mercuric sulphide (cinnabar or vermilion, *Sindur*). Artificial preparation occurs as red crystalline powder and is known as vermilion. (6) Mercurous chloride (calomel). (7) Mercuric nitrite. (8) Mercuric sulphate (white crystalline powder). (9) Sulphate of mercury (lemon-yellow powder). (10) Mercuric cyanide. (11) Ammonio-mercuric chloride. (12) Organic compounds of mercury.

Action: Mercury has affinity for a number of cellular components essential for the function and survival of the cell, such as enzymes, membrane proteins, nucleic and mitotic apparatus.

Fate and Excretion: Mercury is impounded in all tissues, particularly in liver, kidneys, spleen and bones. Excretion is by kidneys, liver and colonic mucous membrane. It is also excreted in the saliva, milk, sweat and faeces.

Symptoms: First phase: Acrid metallic taste and a feeling of constriction in the throat, hoarse voice, difficulty in breathing. The mouth, tongue and fauces become corroded, swollen and show a greyish-white coating. Hot burning pain in the mouth, extending down to the stomach and abdomen, followed by nausea, retching and vomiting. The vomit contains greyish slimy mucoid material with blood and shreds of mucous membrane. This is followed by diarrhoea with bloodstained stools and tenesmus. Circulatory collapse occurs soon. Inhalation of fumes produce nervous symptoms, e.g. ataxia, restriction of visual field, paresis and delirium.

Second phase: If the person survives, second phase begins in one to three days. Glossitis and ulcerative gingivitis appear within 24 to 36 hours. Severe infection, loosening of teeth and necrosis of the jaw may occur. In two to three days, renal tubules show necrosis and produce transient polyuria, albuminuria, cylindruria, uraemia and acidosis. Recovery may occur within 10 to 14 days. After many days, membranous colitis develops and produces dysentery, tenesmus, ulceration of colonic mucosa and haemorrhage.

Levels in blood and urine should not exceed 3.6 µg/L and 15 µg/L respectively. Symptoms may develop when blood and urine levels exceed 20 µg/L and 60 µg/L.

Fatal Dose: One to two grams of mercuric chloride.

Fatal Period: Three to five days.

Treatment: (1) Milk, egg-white, 5% salt-poor albumin, 5% sodium formaldehyde sulphoxylate or 2 to 5% sodium bicarbonate may be used for gastric lavage to bind the mercury. Activated charcoal is of uncertain benefit. (2) Cathartics should not be given but whole bowel irrigation may be beneficial. (3) Penicillamine is used for less severe mercury vapour and inorganic mercury poisoning. It is given orally in a dose of 100 mg/kg every 6 hours (maximum one g/day) for 5 days in acute poisoning in children and 250 mg. four times a day for 5 days in adults. For chronic poisoning in children 25 to 40 mg. maximum one g/day divided into two doses and for adults 250 mg. twice a day is given until urine mercury levels are less than 50 µg/l. (4) BAL is the chelating agent of choice. Dosage regimen is same as for arsenic. (5) BAL and penicillamine in combination should not be used as they may cause formation of a toxic compound. (6) Demulcents. (7) High colonic lavage with 1:1000 solution of sulphoxylate twice daily. (8) Urine must be kept alkaline. (9) EDTA should not be used as it is nephrotoxic. (10) Haemodialysis if there is significant kidney damage. (11) Symptomatic.

Postmortem Appearances: The mucosa of the gastrointestinal tract shows inflammation, congestion, coagulation and corrosion. If the person survives for few days, the large intestine shows necrosis due to the re-excretion of mercury into the large bowel. Acute tubular and glomerular degeneration or haemorrhagic glomerular nephritis is seen. The liver is congested and shows cloudy swelling or fatty change.

Chronic Poisoning (Hydrargyrism): This may result from (1) continuous accidental absorption by the workers, (2) excessive therapeutic use, (3) from recovery from a large dose, and (4) if ointment is used as external application for a long time.

The symptoms are salivation, inflammation of gums and occasionally a blue line at their junction with teeth, sore mouth and throat, loosening of teeth, gastrointestinal disturbances, fine tremors of the tongue, hands, arms and later of legs, anaemia, anorexia, loss of weight and chronic inflammation of kidneys with progressive uraemia. Tremors (sometimes called Danbury tremors) occur first in the hands, then progress to lips and tongue and finally involve arms and legs. The tremor is moderately coarse and is interspersed by jerky movements. The advanced condition is called hatter's shakes or glass-blowers shakes, because they are common in persons working in glass-blowing and hat industries. There may be mental disturbances. Mercurial erethism is

seen in persons working with mercury in mirror manufacturing firms. This term is used to refer to the psychological effects of mercury toxicity. These include anxiety, depression, shyness, timidity, irritability, loss of confidence, mental depression, delusions and hallucinations, or suicidal melancholia, or manic depressive psychosis (mad hatter), emotional instability, loss of memory and insomnia. Mercurialentis is a peculiar eye change due to exposure to the vapour of mercury. It is due to brownish deposit of mercury through the cornea on the anterior lens capsule. Slit-lamp examination demonstrates a malt-brown reflex from the anterior lens capsule. It is bilateral and has no effect on visual acuity.

Acrodynia or pink disease is thought to be hypersensitivity reaction especially in children. This can be caused by chronic mercury exposure in any form. There is irritation of hands and feet usually followed by desquamation, loss of hair, hyperkeratosis and excessive sweating.

Minimata disease is a type of organic mercurial poisoning due to eating of fish poisoned by mercury.

It is introduced into the vagina for producing abortion. Homicidal and suicidal poisoning is rare.

LEAD

Poisonous compounds: It is a heavy steel-grey metal. Metallic lead and all its salts are poisonous. The principal salts are: (1) Lead acetate (sugar of lead): white crystals. (2) Lead carbonate (*safeda*): a white crystalline powder. (3) Lead chromate: a bright-yellow powder. (4) Lead monoxide (litharge): pale brick-red or pale orange masses. (5) Lead tetroxide (red lead; vermilion; *sindur*): scarlet crystalline powder.

ACTION: At the cellular level, lead interacts with sulphydryl groups and interferes with the action of enzymes necessary for haem synthesis and for haemoglobin and cytochrome production. It causes haemolysis.

Fatal Dose: About 20 g. lead acetate: 40 g. lead carbonate.

Fatal Period: One to two days.

ACUTE POISONING: The symptoms are: an astringent and metallic taste, dry throat, thirst, burning abdominal pain, nausea, vomiting, sometimes diarrhoea, peripheral circulatory collapse, headache, insomnia, paraesthesias, depression, coma and death.

TREATMENT: (1) Gastric lavage with one percent solution of sodium or magnesium sulphate. (2) Demulcents. (3) The combination of BAL and calcium disodium versenate is effective. (4) Penicillamine. (5) Calcium chloride 5 mg. of a 10% solution i.v. or calcium gluconate ten ml. of a 10% solution i.v. cause deposition of lead in the skeleton from the blood. (6) Peritoneal or haemodialysis.

Postmortem Appearances: Signs of acute gastro-enteritis are seen.

Chronic Poisoning (plumbism; saturnism): Causes: (1) Inhalation of lead dust and fumes by makers of white lead and makers and users of lead paints (most common source of exposure for children), smelters, plumbers, glass-polishers, printers, enamel workers, glass blowers, etc. (2) Continuous absorption of minute amounts from drinking water stored in lead cisterns, from tinned food contaminated with lead from the solder, and from constant use of hair dyes and cosmetics containing lead. (3) Absorption through raw or intact skin. (4) Use of *ghee* stored in brass or copper vessels lined inside with tin in which oleate of lead is formed and also by taking food cooked in tinned vessels. (5) Absorption of vermilion applied to the scalp. (6) Children can be chronically poisoned through chewing or licking toys, walls, furniture, etc., painted with lead based compounds. Chronic poisoning results from a daily intake of one to two mg. of lead. Lead vapour is more dangerous than dust. Lead is a typical cumulative poison.

Signs and Symptoms: (1) Facial pallor: The facial pallor particularly about the mouth is one of the earliest and most consistent sign. (2) Anaemia: There may be polycythaemia with polychromatophilia in early stages, but later there is anaemia which is associated with polychromasia, punctate basophilia, reticulocytosis, poikilocytosis, anisocytosis, nucleated red cells (sideroblasts) and an increase in mononuclear cells, whereas polymorphonuclear cells and platelets are decreased. Punctate basophilia or basophilic stippling means the presence of many dark-blue coloured pinhead sized spots in the cytoplasm of red blood cells, due to toxic action of lead on porphyrin metabolism. Reticulocytes and basophilic stippled cells result from the inhibition of 5-pyrimidine nucleotidase, an impaired ability to rid the cells of RNA degradation products and the aggregation of ribosomes. Stippled red cells may also be seen in arsenic and zinc poisoning. Eosinophilia

is more common than basophilic stippling. Porphyrins excreted in urine may be 500 µg per day. **(3) Lead line: A stippled blue line, called Burtonian line, is seen on the gums in 50 to 70% of cases.** It appears due to subepithelial deposit of granules at the junction with teeth, only near dirty or carious teeth, within a week of exposure, usually on upper jaw. It is due to formation of lead sulphide by the action of hydrogen sulphide formed by decomposed food in the mouth. A similar **blue line may be seen in cases of poisoning by mercury, copper, bismuth, iron and silver. (4) Colic and constipation:** It is usually later symptom. Colic of intestines, ureters, uterus and blood vessels occur in 85% of cases. The colic occurs at night and the pain may be very severe. Individual attacks last only a few minutes, but may recur for several days or weeks. Constipation is usual but diarrhoea or vomiting may occur. **(5) Lead palsy:** It usually occurs late and is seen in less than 10% of cases. There may be tremors, numbness, hyperaesthesia, and cramps before the actual muscle weakness. It is commoner in adults than in children, and men are particularly affected. The muscle groups affected are those most prone to fatigue. **Usually, the extensor muscles of the wrist (wrist drop due to involvement of radial nerve) are affected, but rarely muscles of eye or intrinsic muscles of hand or foot are affected.** The paralysis is associated with degeneration of the nerve and atrophy of the muscles. Recovery may be complete but is usually slow. **(6) Encephalopathy: Lead encephalopathy in some form is said to be present in almost every case of plumbism.** It is common in children. The symptoms are vomiting, headache, insomnia, visual disturbances, irritability, restlessness, delirium, hallucinations, convulsions, coma and death. It is usually irreversible and about 85% have permanent brain damage. Death occurs in about 25% of cases. **(7) Cardiovascular system and kidneys:** Lead causes vascular constriction, leading to hypertension and permanent arteriolar degeneration. Chronic arteriosclerotic nephritis and interstitial nephritis occur. **(8) Reproductive system:** Menstrual derangements, such as amenorrhoea, dysmenorrhoea, menorrhagia, sterility of both sexes and abortion (between 3 and 6 months) are frequent. **(9) Other systems:** They are dyspepsia, emaciation, irritability, headache, vertigo, loss of hair and drowsiness.

Diagnosis: (1) History. (2) Clinical features. (3) X-ray evidence of increased radio-opaque bands or lines at the metaphyses of long bones and along margins of iliac crest is seen in children. (4) Erythrocyte protoporphyrin (EP) commonly assayed as zinc protoporphyrin (ZPP) are usually below 35 µg/L is not sufficiently sensitive at lower blood lead levels. (5) Basophilic stippling. (6) X-ray may show radio-opaque material in the G.I. tract, if lead is ingested during preceding 36 to 48 hours.

Treatment: (A) Severe acute poisoning with encephalopathy: (1) BAL 4 mg/kg immediately (in children). Repeat the same dose at 4 hourly intervals until blood lead levels fall below 40 µg/100 ml. Then reduce BAL to 12 mg/kg/day in 3 divided doses. (2) $CaNa_2$ EDTA 75 mg/kg/day i.v. infusion. Reduce EDTA to 50 mg/kg/day as condition improves. (3) The above regimen is continued until patient is asymptomatic and can tolerate oral chelation with D-penicillamine 10 mg/kg/day or DMSA, 10 mg/kg/day t.i.d. for 20 days.

(B) Severe poisoning without encephalopathy: (BL more than 70 µg/100 ml): (1) BAL 12 mg/kg/day. (2) EDTA 50 mg/kg/day. (3) Discontinue BAL when blood level falls below 40 µg/100 ml but continue EDTA for 5 more days. (4) Continue oral chelation until the BL falls below 15 µg/100 ml or for 3 months.

(C) Moderate poisoning (BL. between 45 to 75 µg/100 ml): (1) EDTA 50 mg/kg/day. (2) When BL level falls below 40 µg/100 ml begin oral chelation.

(D) Mild poisoning (BL. 20 to 35 µg/100 ml): D-penicillamine 30 mg/kg/day in 3 divided doses. Start with one-fourth of the calculated dose. Double this after one week. Double again after one week. Continue this until BL falls to less than 15 µg/100 ml. or for 3 months.

DMSA (succimer) 10 mg/kg/day t.i.d. for 20 days is more effective and less toxic.

Postmortem appearances are not constant. The stomach and intestines may show ulcerative or haemorrhagic changes and are contracted and thickened. The liver and kidneys are contracted. The heart may be hypertrophied and there may be atheroma of the aorta and aortic valves. Bone marrow shows hyperplasia of leucoblasts and erythroblasts with a decrease in fat cells.

Absorption: Absorption of inorganic lead compounds from digestive tract is slow. Dusts or fumes are rarely absorbed by the lungs. Tetraethyl lead and other alkylated compounds are absorbed from the skin. Adults may consume up to 300 mg. of lead every day but only about 10% of this is absorbed. Absorption is greater and more rapid by inhalation. Children absorb 50% of lead. Bullets or pellets lodged in joints, peritoneum, pericardium or other soft tissues may result in lead poisoning.

Distribution: Lead is normally present in almost all tissues. It is a typical cumulative poison. The bones contain large amounts and also hair and nails. Lead crosses the placenta. It is stored in the bones as phosphate and carbonate. High calcium level favours storage, while calcium deficiency causes lead to be released into blood stream.

Excretion: Lead is excreted largely in faeces. Absorbed lead is excreted mostly in urine.

Poisoning: (1) Acute poisoning is very rare. (2) Chronic poisoning is more common and is regarded as an industrial disease. (3) Accidental chronic poisoning occurs in workers with the metal. (4) Diachylon paste (lead oleate) or red lead is used locally for procuring abortion. (5) Red lead is sometimes used alone or mixed with arsenic as a cattle poison.

COPPER

Copper as a metal is not poisonous. **Copper compounds are powerful inhibitor of enzymes.**

Poisonous Compounds: (1) Copper sulphate (blue vitriol) occurs in large, blue crystals. (2) Copper subacetate (verdigris), occurs in bluish-green masses or powder.

Signs and Symptoms: Symptoms appear in 15 to 30 minutes. There is a metallic taste, increased salivation, burning pain in the stomach with colicky abdominal pain, thirst, nausea, eructations and repeated vomiting. The vomited matter is blue or green. There is diarrhoea with much straining; motions are liquid and brown but not bloody. Oliguria, haematuria, albuminuria and uraemia may occur. In severe cases, jaundice and cramps of legs or spasms and convulsions occur. The breathing is difficult, cold perspiration and severe headache occur. In some cases, paralysis of limbs is followed by drowsiness, insensibility, coma and death.

Fatal Dose: Copper sulphate: 30 g.; Copper subacetate, 15 g.

Fatal Period: One to three days.

Treatment: (1) Stomach wash with **one percent solution of potassium ferrocyanide forms an insoluble cupric ferrocyanide**. (2) N-penicillamine is very effective. (3) E.D.T.A. or B.A.L. (4) Demulcent drinks form insoluble albuminate of copper. (5) Haemodialysis is useful in the early stage of poisoning.

Postmortem Appearances: The skin may be yellow. Greenish-blue froth may be present at mouth and nostrils. The gastric mucosa and stomach contents are greenish or bluish. The gastric mucosa may be congested, swollen, inflamed, and occasionally eroded. The liver may be soft and fatty.

Absorption and Excretion: Copper is a normal constituent of the body. It is absorbed through the lungs, mucous membranes and raw surfaces. It is eliminated more by the bowels than by the kidneys and in traces in saliva, bile and milk.

Poisoning: (1) It is rarely used for suicide or homicide because of the colour and taste. (2) Accidental poisoning results from eating food contaminated with verdigris which is formed from the action of vegetable acids on copper cooking vessels which are not properly tinned on the inside. Ingestion of food to which copper has been added to keep the green colour of the vegetables may cause poisoning. Children sometimes swallow copper sulphate attracted by its colour. (3) Sometimes, the salts are taken internally for abortion. (4) Rarely, it is used as a cattle poison.

IRON

SYMPTOMS: They are divided into four stages. Few hours after ingestion, vomiting, abdominal pain and haemorrhagic gastroenteritis, shock, acidosis and coma occur. The second stage sets in 6 to 24 hours in which patient is symptom free. In the third stage (24 to 48 hours) metabolic acidosis, jaundice, hypoglycaemia, shock, coma with hepatic and renal failure occurs. In the fourth stage, after one to two weeks, late complications such as gastric stricture and pyloric stenosis occur.

Fatal Dose: 20 to 30 g.

Fatal Period: 24 to 30 hours.

TREATMENT: (1) Stomach wash with 5% sodium bicarbonate solution. Instil 5 to 10 g. of desferrioxamine at the end of lavage. (2) Give plenty of egg and milk to

form iron-protein complexes. (3) Desferrioxamine 1 g. i.m. followed by 500 mg. fourth hourly for two doses and finally 500 mg. four to 12 hourly up to a maximum of 6 g. in 24 hours. It can also be given in infusion 15 mg/kg/hr in normal saline. Haemodialysis or exchange transfusion in severe cases.

P.M. APPEARANCES: Gastric mucosa shows haemorrhagic necrosis. The liver may show acute hepatic necrosis and the kidneys degenerative tubular changes.

THALLIUM

Thallium sulphate and thallium acetate are important salts, which are odourless, tasteless and water-soluble. They are used in the dye and glass industries and as rodenticides and insecticides.

SYMPTOMS: They occur after one to 12 days after a therapeutic dose due to overdose or idiosyncracy. In mild cases joint pains in legs and feet, loss of appetite, stomatitis, and drowsiness occur, which pass off in a few days. In severe cases, there is colic, vomiting, pain in muscles, joints and nerves, lethargy, motor and sensory neuropathies, delirium and coma. Hair loss occurs after one to two weeks. Large tufts tend to come away. Loss of outer third of eyebrows is said to be very significant. There may be cardiac manifestations. Maculo-papular skin eruption having butterfly distribution on face is characteristic. There may be Mees lines on the nails. Death occurs due to respiratory failure due to paralysis of respiratory muscles. After recovery, the patient may suffer from peripheral neuritis resembling Guillain-Barre polyneuritis.

Fatal Dose: One g.

Fatal Period: 24 to 36 hours.

TREATMENT: (1) Stomach wash with one percent potassium iodide or Prussian blue. (2) Activated charcoal. (3) Prussian blue (potassium ferric cyanoferrate) 250 mg/kg/day orally in two divided doses. (4) Haemodialysis along with haemoperfusion. (5) Forced diuresis.

P.M. APPEARANCES: Signs of asphyxia are present. Mucosa of stomach may be inflamed with submucous petechial haemorrhages. Alopecia, stomatitis, fatty heart and liver, renal damage, pulmonary and cerebral oedema are seen.

POTASSIUM PERMANGANATE

It occurs as dark-purple, slender crystals, and has a sweet astringent taste. It is a powerful oxidising agent and is used as a disinfectant.

ACTION: In strong solution or in the solid state, it acts as a corrosive or strong irritant, while after absorption, it causes paralysis of the heart.

SIGNS AND SYMPTOMS: Burning pain from the mouth to the stomach, nausea, vomiting, intense thirst, difficulty in speaking or swallowing, dyspnoea, stridor and a persistent spasmodic cough. The lips, gums, teeth, tongue, tonsils and pharynx are discoloured, inflamed and superficially corroded. Shock is sometimes severe and there may be immediate collapse.

Fatal Dose: 5 to 10 g.

Fatal Period: Few hours.

TREATMENT: (1) Gastric lavage should be continued until the washings are colourless; 20% sodium thiosulphate should be used for the initial washings. (2) Demulcents. (3) Activated charcoal.

POISONING: It is sometimes taken orally as an abortifacient. It is rarely used to produce fictitious injuries, e.g. to simulate the lesions of tertiary syphilis by applying a tablet to the skin for 10 to 20 minutes. Sometimes, a tablet is inserted into the vagina to procure abortion.

METAL FUME FEVER: It is caused by inhalation of fumes of zinc, copper, magnesium, nickle, mercury, lead and other heavy metals. The syndrome resembles a flu-like illness which starts 6 to 8 hours after exposure to fumes with fever, chills, cough, dyspnoea, cyanosis, myalgia, salivation, sweating and tachycardia. Symptoms subside in 36 hours after stoppage of exposure.

CHAPTER

27 Inorganic Irritant Poisons

PHOSPHORUS

Varieties: There are two varieties: (1) White or crystalline. (2) Red or amorphous (Table 27-1). It is used in insecticides, rodenticides, fertilisers, incendiary bombs, smoke screens, fireworks, etc.

Action: **It is protoplasmic poison, which affects cellular oxidation.**

Signs and Symptoms: **(1) Fulminating Poisoning:** This is seen when a dose exceeding one gram is taken. These patients usually die within 12 hours due to shock and cardiovascular collapse.

(2) Acute Poisoning: **(A) First Stage:** Symptoms occur within a few minutes to a few hours after exposure and last from 8 hours to 3 days. Ingestion produces burning pain in the throat and abdomen, with intense thirst, nausea, vomiting, diarrhoea and severe abdominal pain. Breath and excreta have garlic-like odour. Luminescent vomit and faeces are diagnostic. Skin contact produces painful penetrating second and third degree burns, which heal slowly.

(B) Second Stage: This is a symptom-free period lasting for 2 to 3 days.

(3) Third Stage: Symptoms of systemic toxicity occur from absorbed poison. There is nausea, prolonged vomiting, diarrhoea, haematemesis, liver tenderness and enlargement, jaundice, and pruritus. Haemorrhages occur into skin, mucous membrane and viscera, due to injury of blood vessels and inhibition of blood clotting. Renal damage results in oliguria, haematuria, casts, albuminuria and sometimes anuria. Involvement of CNS causes convulsions, delirium and coma. If the patient survives, symptoms may persist for a long time. Death may result from shock, hepatic failure, CNS damage, haematemesis or renal insufficiency.

Fatal Dose: 60 to 120 mg.

Fatal Period: 2 to 8 days.

Treatment: (1) Gastric lavage using 1:5000 solution of **potassium permanganate, oxidises phosphorus** into phosphoric acid and phosphates, which are harmless. (2) Medicinal charcoal adsorbs the poison. (3) Stomach can be washed with 0.2% **copper sulphate solution** or 0.2 g. of copper sulphate may be given every 5 minutes until vomiting occurs. **It coats the particles of phosphorus with a film of copper sulphide which is relatively harmless.** (4) **Vitamin K 20 mg. i.v.** in repeated doses to counteract hypoprothrombinaemia or blood transfusion. (5) The bowel should be evacuated with magnesium sulphate.

Table (27-1). Difference between white and red phosphorus

Trait	White phosphorus	Red phosphorus
(1) Colour:	White or yellow.	Reddish-brown.
(2) Appearance:	Translucent, waxy cylinders.	Amorphous, solid mass.
(3) Smell:	Garlicky.	Odourless.
(4) Taste:	Garlicky.	Tasteless.
(5) Luminosity:	Luminous in the dark.	Non-luminous.
(6) Exposure to air:	Oxidises and emits white fumes; ignites at 34°C and as such kept under water.	Non-oxidised, non-fuming, non-inflammable.
(7) Use:	Fertilisers, vermin-killers, rodenticide, fireworks, gunpowder, etc.	On the sides of match box.
(8) Toxicity:	Highly toxic.	Non-toxic.

(6) Wash out the bowel and repeat at intervals for several days. (7) ~~Oils and fats~~ should be avoided as they dissolve phosphorus and promote absorption. (8) If renal failure is severe, peritoneal or haemodialysis may be required. (9) Burns should be thoroughly washed with one percent copper sulphate solution in water.

Postmortem Appearances: There may be no obvious changes in fulminating poisoning. In acute poisoning the body usually shows **signs of jaundice. The gastric and intestinal contents may smell of garlic and may be luminous.** The mucous membranes of the stomach and intestine are yellowish or greyish-white in colour, and are softened, thickened, inflamed and corroded or destroyed in patches. **Multiple smaller or larger haemorrhages are seen in the skin,** subcutaneous tissues, muscles, and serosal and mucosal membranes. The liver becomes swollen, yellow, soft, fatty, and is easily ruptured and shows marbled appearance. Small haemorrhages may be seen on the surface and in the substance with some cellular necrosis. **In persons who survive for a week or longer, the appearances of acute yellow atrophy are present.** The kidneys are large, greasy, yellow and show haemorrhages on the surface. The heart is flabby, pale and shows fatty degeneration. Fat emboli may be found in the pulmonary arterioles and capillaries. The blood may appear tarry and its coagulability is diminished.

Chronic Poisoning: The frequent inhalation of fumes over a period of years causes necrosis of the lower jaw in the region of a decayed tooth. At first there is toothache, which is followed by swelling of the jaw, loosening of the teeth, necrosis of gums, and sequestration of bone in the mandible. **This condition is known as phossy jaw, in which osteomyelitis and necrosis of the jaw occurs, with multiple sinuses discharging foul-smelling pus.**

The Circumstances of Poisoning: (1) Accidental poisoning in children may occur due to chewing of fireworks or by eating rat paste. (2) Phosphorus is occasionally used for homicide mixed with alcohol, coffee, etc. (3) It is taken by mouth or introduced into the vagina to produce abortion. (4) For arson white phosphorus covered with dung or wet cloth is thrown on huts. When the covering becomes dry, the roof catches fire.

IODINE

It occurs as bluish-black, soft and scaly crystals and has a metallic lustre and an unpleasant taste. It gives off a violet-coloured vapour at all temperatures, which has a characteristic odour.

ACTION: It is a protoplasmic poison fixing protein and causing necrosis. Vapours irritate respiratory passages.

SIGNS AND SYMPTOMS: Inhalation produces glottic and pulmonary oedema. Swallowed in the solid form, it acts as an acid corrosive poison. The lips and the angles of the mouth are stained brown. The vomited matter and excreta are dark-yellow or blue in colour, contain blood and have the peculiar odour of iodine. The urine is scanty or suppressed, red-brown in colour, contains albumen. Nephritis and renal failure occur. Pulse is slow and weak, skin cold and clammy and there may be skin rashes.

FATAL DOSE: 2 to 4 g (30 to 60 ml. of tincture).

FATAL PERIOD: 24 hours.

TREATMENT: Evacuate the stomach by emetics or wash it out with warm water containing soluble starch and albumen or 5% solution of sodium thiosulphate. Give alkalis, arrow root, and barley water and treat symptomatically. Skin lesions can be treated with 20% alcohol.

POSTMORTEM APPEARANCES: The mucosa of the stomach and intestines is inflamed, excoriated and may be brown. The heart, liver and kidney may show fatty degeneration. There may be oedema of the brain.

CHRONIC POISONING (IODISM): The symptoms are pain over the frontal sinus, running of the nose, conjunctivitis, bronchial catarrh, salivation, nausea, vomiting, purging, emaciation, wasting of breasts, testes, etc., and acne or erythematous patches on the skin.

CHLORINE

It is a greenish-yellow gas having an unpleasant irritating odour. It is an active oxidising agent and causes extensive destruction of organic tissue.

SIGNS AND SYMPTOMS: When inhaled, it has an irritant and suffocative effect. The symptoms of acute poisoning are intense irritation of the eyes and throat and mucous membrane of respiratory passages, with violent coughing and extreme dyspnoea, lachrymation, rhinorrhoea, laryngeal and pulmonary edema, respiratory failure, nausea, vomiting and spasm of the glottis. Death is caused by cardiac failure.

POSTMORTEM APPEARANCES: These are mainly asphyxial. There is inflammation of the respiratory tract, pulmonary oedema, rupture of alveolar walls, haemorrhages and thrombosis in the lung beds and increased viscosity of the blood.

IRRITANT MECHANICAL POISONS

They are not absorbed into the body, but they act mechanically and have only local action, and cause irritation of GIT when swallowed. Powdered glass, diamond powder, pins, needles, nails, chopped animal and vegetable hair are the examples.

POWDERED GLASS: Symptoms: There is sharp burning pain in the throat, stomach and intestines, nausea, vomiting, usually constipation but rarely diarrhoea. Death may result from shock if stomach or the intestine is perforated.

Treatment: Give bulky food and later emetics and purgatives.

Postmortem Appearances: Erosions may be seen extending from the mouth to the upper part of small intestine. The mucosa of the stomach is covered with sticky mucus and the glass particles may be found in the stomach and intestines. The mucosa of the GIT is congested and may be inflamed.

Medicolegal Importance: It is rarely used for suicide or homicide. It is taken in powdered form mixed with some articles of food. Occasionally, it is used as a cattle poison.

CHAPTER

28 Organic Irritant Poisons

RICINUS COMMUNIS

Characteristics: The castor plant *(arandi)* grows all over India. Seeds are variable, smooth, flattened-oval, mottled, light and dark brown, or white with yellow-brown or gray markings, or wholly black or red. They are of two sizes, big and small. The small seeds are about 1.2 cm. long and 0.8 cm. broad and resemble croton seeds (Plate 23-134 and 24-147).

Active principles: Entire plant is poisonous, but **seeds are more poisonous, containing toxalbumen ricin,** a water-soluble glycoprotein (highest level in the seeds) and a powerful allergen. The seeds are rich in a purgative oil, which is pale-yellow with a faint odour and acrid taste.

Toxalbumen: A toxalbumen or phytotoxin is a toxic protein, which resembles a bacterial toxin in action, and causes agglutination of red cells with some haemolysis and is antigenic. Phytotoxin is a toxin produced by a plant. Ricin, crotin and abrin are phytotoxins. Animal toxalbumins are snake and scorpion venoms.

The 'press cake' contains ricin and is poisonous, whereas castor oil contains ricinoleic acid but not ~~toxalbumen~~, and **is not poisonous**, as it does not contain ~~ricin~~. The unbroken seeds are non-poisonous when swallowed and also when cooked. **Ricin is said to be 6000 times more powerful than cyanide.** It can be absorbed through inhalation, ingestion, injection and through skin contact.

Signs and Symptoms: Symptoms include burning in mouth, throat and stomach, salivation, nausea, vomiting, bloody diarrhoea, abdominal pain, haemorrhagic gastritis, thirst, impaired sight, weak, rapid pulse, cramps in calves and abdominal muscles, drowsiness, delirium, convulsions, shallow breathing, uraemia and jaundice, dehydration, collapse and death. **Ricin is excreted by the intestinal epithelium.**

Fatal Dose: 5 to 10 seeds; ricin 50 to 100 µg

Fatal Period: 2 to 5 days.

Treatment: Gastric lavage, demulcents, symptomatic.

Postmortem Appearances: The mucosa of the GIT is congested, softened and inflamed with occasional erosions and submucous haemorrhages. Ricin produces haemorrhagic inflammation of the GIT even when given subcutaneously. There may be haemorrhages in the internal organs.

Poisoning: Accidental poisoning may occur in children from eating the seeds. Rarely, the powdered seeds are given for ~~homicide~~. The powder of seeds causes conjunctivitis when applied to the eye. Its use by terrorists might involve poisoning of water or food, inoculation via ricin-laden projectiles or aerolisation of liquid ricin, or distribution of powder.

CROTON TIGLIUM

Characteristics: Croton tiglium *(jamalgota or naepala)* seeds contain crotin, a toxalbumen which is not expressed with the oil. The oil is brown, viscid, has unpleasant odour and acrid, burning taste. Seeds are oval, dark-brown with longitudinal lines (Plate 23-135). They have no ~~smell~~.

Active principles: Crotonoside, a glycoside, which is less poisonous, is also present. The oil contains a powerful vesicating resin composed of crotonoleic acid, methyl crotonic acid and several other fatty acids.

Signs and Symptoms: There is hot burning pain from mouth to stomach, salivation, vomiting, purging, vertigo, prostration, collapse and death. Applied to the skin, the oil produces burning, redness and vesication.

Fatal Dose: 4 to 5 seeds; 1 to 2 ml. of oil.

Fatal Period: Six hours to three days.

Treatment: Stomach wash, demulcent drinks and symptomatic.

Postmortem Appearances: There is congestion, inflammation and erosion of the mucosa of stomach and intestines and congestion of internal organs.

Poisoning: (1) Accidental poisoning results from swallowing croton oil by mistake or by eating seeds or inhaling their dust. (2) ~~Suicide~~ and ~~homicide~~ are rare.

(3) The root and oil are sometimes taken internally as an abortifacient. (4) Oil is used as arrow poison.

ABRUS PRECATORIUS

Characteristics: It is also known as jequirity, rosary bead, or Indian liquorice *(gunchi or rati)*. It is a slender, twining, climbing plant found all over India. The seeds are egg-shaped, bright scarlet colour with a large black spot at one end, 8 mm. long and 6 mm. broad, and weigh 105 mg. on an average. Seeds may be white with black spot, all black, yellow or blue (Plate 23-136).

Active principles: **The seeds contain an active principle abrin, a toxalbumen, which is similar to viperine snake venom; also present are abrine** (amino acid) **haemagglutinin in the cotyledons; a lipolytic enzyme, and abralin** (glycoside). Abrin inhibits protein synthesis and causes cell death. All parts of the plant are poisonous. Seeds are tasteless and odourless. Abrin inhibits protein synthesis and causes cell death.

Signs and Symptoms: Symptoms after ingestion may be delayed from a few hours to two or three days. They include abdominal pain, nausea, vomiting, diarrhoea, weakness, cold perspiration, trembling of the hands, weak rapid pulse and rectal bleeding. When an extract of seeds is injected under the skin of the animal, inflammation, oedema, oozing of haemorrhagic fluid from the site of puncture, and sometimes necrosis occurs surrounding the site of injection. The animal does not take food and drops down after 3 to 4 days and cannot move. Tetanic convulsions occur or the animal becomes cold, drowsy or comatose and dies. **The symptoms resemble those of viperine snake bite,** and as such poisoning is not suspected by the farmer.

In man, at the site of injection, painful swelling and ecchymosis develops, with inflammation and necrosis. There is faintness, vertigo, vomiting, dyspnoea, and general prostration. Convulsions may occur before death from cardiac failure.

Fatal Dose: 90 to 120 mg. by injection or one to two seeds. Subcutaneously, abrin is 100 times as toxic as by the oral route.

Fatal Period: 3 to 5 days.

Treatment: Injection of anti-abrin. The needle should be dissected out.

Poisoning: (1) **The seeds are used for killing cattle and rarely for homicide**. The cattle are poisoned by leather workers to obtain hides cheaply or for revenge. **The seeds are decorticated and alone or mixed with datura, opium and onion are made into paste with spirit and water, and small sharp-pointed spikes or needles or '*suis*' are prepared which are then dried in the sun. The needles are 15 mm. long and weigh 90 to 120 mg. Two needles are inserted by their base into holes in a wooden handle. A blow is struck to the animal with great force which drives the needle into the flesh.** For homicide, the needle is kept between two fingers, and the person is slapped which drives the needle into the body. (2) Powdered seeds are used by malingerers to produce conjunctivitis. (3) The seeds are used as an abortifacient and as arrow poison.

ERGOT

Ergot is the dried sclerotinum of the fungus Claviceps purpurea, which grows on cereals like rye, barley, wheat, oats, etc. It gradually replaces the grain forming a curved, dark-purple or black compact mass, and has disagreeable taste. It contains about thirty alkaloids, but ergotoxine, ergotamine and ergometrine are important.

SIGNS AND SYMPTOMS: In acute cases there is nausea, vomiting, diarrhoea, giddiness, tightness in the chest, difficulty in breathing, marked muscular weakness and exhaustion. There may be tingling and numbness in the hands and feet, paraesthesias, followed by twitching or cramps in the muscles. Bleeding from the nose and other mucous surfaces is common after large doses. The pulse is rapid and weak, pupils are dilated with dimness of vision, blood pressure is raised.

ST. ANTHONY'S FIRE is epidemic of dysesthesia (burning sensation in extremities, delirium and convulsions) produced by eating rye bread contaminated with ergot.

CHRONIC POISONING (ergotism): There is tingling and numbness of the skin, vasomotor disturbances leading to dry gangrene of the fingers, toes, ears, nose, etc. There is a sensation of insects creeping under the skin. Neurological disorder characterised by hallucinations, ataxia and convulsions may occur.

FATAL DOSE: 5 to 10 g.

FATAL PERIOD: One day.

TREATMENT: Wash the stomach and give purgatives. Vasodilators, e.g., nitrites are useful.

POSTMORTEM APPEARANCES: They are not characteristic.

POISONING: The consumption of bread made with contaminated rye is the chief cause of ergotism. Ergot is commonly used as an abortifacient.

CAPSICUM ANNUM

Capsicum or chillies (*mirch*) have a pungent odour and taste and are used as a condiment (Plate 23-138). **They are not fatal. The active principles are capsaicin (alkaloid) and capsicin.**

Signs and Symptoms: In large doses, it acts as an irritant poison and causes difficulty in swallowing, pain in the stomach and inflammation of oesophagus and stomach. Applied to the skin or mucosa it causes irritation. Powder thrown in the eyes causes severe irritation leading to lachrymation, burning pain and redness.

Treatment: (1) Bathe affected skin in vinegar or ice-cold water. (2) If ingested, sucking of ice cubes, or sips of ice cold water.

Criminal Uses: (1) Powder is thrown into the eyes to facilitate robbery. (2) When theft or confession of some guilt has to be obtained, the person is tortured by introducing the powder into vagina, rectum or urethra or by rubbing on the female breasts.

SEMECARPUS ANACARDIUM

Characteristics: Marking nuts (bhilawa) are black, heart-shaped with rough projection at the base. They have a thick, cellular pericarp, which contains an irritant juice which is brownish, oily and acrid but turns black on exposure to air (Plate 23-140). The active principles are semecarpol (0.1%) and bhilawanol (15 to 17%).

Signs and Symptoms: Applied externally, the juice causes irritation and a painful blister which contains **acrid serum which produces eczematous eruptions of the neighbouring skin with which it comes into contact and there is itching. The lesion resembles a bruise.** Later, an ulcer is produced and there may be sloughing.

Taken by mouth, the juice causes less irritation. In large dose, it produces blisters on throat and severe gastrointestinal irritation, dyspnoea, tachycardia, hypotension, cyanosis, areflexia, delirium, coma and death.

Fatal Dose: 5 to 10 g.

Fatal Period: 12 to 24 hours.

Treatment: (1) Gastric lavage. (2) Demulcent drinks. (3) When applied externally wash with lukewarm water containing antiseptic.

Postmortem Appearances: Blisters are seen in the mouth, throat and stomach which are congested and inflamed.

Circumstances of Poisoning: (1) Accidental poisoning may result from the administration of juice internally by quacks. (2) Homicidal and suicidal poisoning is rare. (3) Sometimes, the juice is introduced into the vagina as a punishment for infidelity. (4) For criminal abortion, the bruised nut is applied to the cervical os. (5) To support a false charge of assault, the juice is applied to skin which produces lesions simulating bruises. (6) The juice may be thrown on the body to cause injury. (7) Malingerers use the juice to produce ophthalmia.

CALOTROPIS

Characteristics: Calotropis gigantea (*akdo, madar*) has purple flowers and Calotropis procera has white flowers. They grow wild throughout India (Plate 24-143).

Active principles: The active principles are uscharin, calotoxin, calactin and calotropin. The leaves and stem when incised yield thick milky juice.

Signs and Symptoms: Applied to the skin, it causes redness and vesication. When taken by mouth, the juice produces an acrid bitter taste and burning pain in throat and stomach, salivation, stomatitis, vomiting, diarrhoea, dilated pupils, tetanic convulsions, collapse and death.

Fatal Dose: Uncertain.

Fatal Period: 6 to 12 hours.

Treatment: Stomach wash, demulcents and symptomatic.

Postmortem Appearances: Inflammation of GIT is seen. The abdominal viscera and brain are congested.

Circumstances of Poisoning: (1) It is sometimes used for infanticide and rarely for suicide or homicide. (2) The juice is taken by mouth or introduced into uterus on an abortion stick for criminal abortion. (3) As a cattle poison, it is smeared on a cloth and pushed into the rectum of the animal or is given with fodder. (4) It is sometimes used as arrow poison. (5) Occasionally, to produce artificial bruise. (6) The flowers, leaves, root and juice are used in Indian medicine. (7) Juice is used as a vesicant, depilatory and for chronic skin infections. (8) **The root of calotropis procera is highly poisonous to cobras,**

and other poisonous snakes, which cannot stand even its smell.

PLUMBAGO ROSEA (*lal chitra*)

PLUMBAGO ZEYLANICA (*chitra*)

The root contains as an active principle, plumbagin, a crystalline glycoside.

Fatal Dose: Uncertain.

Fatal Period: Few days.

Symptoms: Applied externally roots produce irritation and blisters. Taken internally, there is burning pain from mouth to stomach, vomiting, thirst, diarrhoea, collapse and death.

Poisoning: (1) Roots are rarely used for homicide. (2) Roots are taken by mouth or applied as paste to the cervix or to abortion stick for criminal abortion. (3) Sometimes, the paste made from the roots is applied to the skin to simulate a bruise.

CANTHARIDES

The Spanish fly (blister beetle) is 2 cm. long and 0.6 cm. broad (Plate 19-113). The powder of the dried body is greyish-brown and contains shiny green particles. **The active principle is cantharidin.** It is used externally as an irritant. It contains about 3% cantharidin. **Cantharidin is readily absorbed from all surfaces including the skin.**

Symptoms: Applied to the skin, redness and burning pain are produced after 2 to 3 hours followed by vesication. Taken internally, there is burning sensation in the mouth and throat, followed by pain in the stomach, nausea and vomiting of bloody mucus, severe thirst, difficulty in swallowing and speech. Later a dull pain is felt in the loins, the urine is scanty and blood-stained. Priapism may occur; there is often tenesmus. Abortion occurs in pregnant women.

Fatal Dose: 15 to 50 mg. of cantharidin, or 2 to 3 g. of powdered cantharides.

Fatal Period: 24 to 36 hours.

Postmortem Appearances: The mouth, stomach and intestinal canal show inflammation and sometimes vesication. The kidneys are acutely inflamed and there is haemorrhage in the renal pelvis and bladder. The bladder mucosa is inflamed and ecchymoses may be present. The surfaces of the heart and endocardium show haemorrhages. The lungs may be oedematous and the air-passages contain blood-stained mucus.

Poisoning: (1) Accidental poisoning may occur by its external application as counterirritant. (2) Rarely used for homicide. (3) Criminal abortion. (4) It is used as an aphrodisiac.

SNAKES

There are more than 3500 species of snakes, but only about 250 are venomous. In India about 216 species are found, of which 52 are poisonous. Only five are dangerously poisonous to man; king cobra, cobra, common krait, Russell's viper and saw-scaled viper. The most common poisonous snake is common krait.

CLASSIFICATION: The poisonous snakes may be divided into five families. (1) (A) Viperidae: Russell's viper, gaboon viper, sawscaled viper, puff adder. (B) Cortalidae: rattlesnakes, pigmy rattlesnakes, copper heads, cotton mouths (water moccasins), pit viper, and the massasaugas, bushmaster. (2) Elapidae: Cobras, kraits, mambas, tiger snake, taipan, death adder, copperhead snakes, coral snakes. (3) Hydrophidae: The sea snakes. All sea snakes are poisonous but they seldom bite. (4) Colubridae: Boom slangs, bird snake of the African continent (5) Atractaspididae: African and Middle Eastern burrowing asps or stiletto snakes also known as burrowing or mole vipers or adders, or false vipers.

COMMON POISONOUS SNAKES: (1) The COBRA (*nag*) has a hood which on dorsal side often bears a double or single spectacle mark, but it has sometimes an oval spot surrounded by an ellipse. Head scales are large and the third labial touches the eye and the nasal shield. There are two black spots and three black bands on the underside of the hood. There is a white band in the region where the hood touches the body. The colour is brown or dark. It grows to a length of about 2 metres (Plate 22-127). (2) The KING COBRA (*rajnag, nagraj*) has a hood but no mark on it, and the length is about 3 to 4 metres. The colour may be yellow, green, brown or black and has yellowish or white cross-bands in the body. The tail scales are entirely present in their proximal ends, but divided in the distal end. (3) The COMMON KRAIT (*karayat*) is steel-blue, often shining and has single or double white bands across the back and a creamy white belly. Its length is one to one-and-half metres. The stripes are not very distinct in the anterior region. The head is covered with large shields. The tail is round. These snakes are nocturnal in habit (Plate 22-128). (4) The BANDED KRAIT (*ahiraj, raj sanp*) is one-and-half to two metres in length. The tail ends bluntly and is swollen at the tip. It has a jet black five cm. wide cross-band, alternating with a deep-yellow band of the same size on its back. There is a black mark

on the neck which is spread up to the eyes (Plate 22-129). (5) RUSSELL'S VIPER, or daboia (*kander*) has a flat, heavy and triangular head with a white V-shaped mark, the angle of the V pointing forwards. It has three rows of diamond-shaped black or brown spots along the back, the outer two rows consisting of spots ringed with white edges. Its body is whitish with dark semilunar spots. It is one-and-half metres long. It is heard to hiss loudly and continuously (Table 28-2) and (Plate 22-131). (6) The SAW-SCALED VIPER (echis carinatus; *afai*) is brown, half metre in length and has wavy white line on each flank of the back with diamond-shaped areas between these two lines. It has a triangular head, the upper surface of which is covered with a white mark resembling a bird's footprint or an arrow. The tail is short and tapering. The broad belly plates with brown or dark spots, small scales on the head and entire shields beneath the tail are the distinguishing features (Table 28-1) and (Plate 22-130). Pit vipers have a pit located between the nostrils and the eyes.

Common krait, cobra, Russell's viper, and saw-scaled viper are seen all over India. Banded krait is seen mainly in East India, and king cobra in Western Ghats.

Sea Snakes: Twenty types are seen in Indian waters, all of them being poisonous. They are black, greenish-black or bluish-black with or without bands.

Fangs: All the poisonous snakes have two fangs. These are curved teeth situated on the maxillary bones and lie along the jaws and are covered by a flap of mucous membrane. When the snake is about to bite, they become erect and point directly forward. They are bigger than the other teeth and are grooved or canalised in poisonous snakes, and are solid in non-poisonous snakes (Table 28-2). When a poisonous snake bites, it normally leaves two deep faint impressions, the distance between them being 8 mm to 4 cm. A side-swipe may produce a single puncture and also small marks of other teeth. Non-poisonous snake bites leave a number of small impressions in a row.

Even a thin layer of clothing may afford great protection, as the venom is injected superficially by vipers, and also by elapids and hydrophids. About 20% of patients bitten by poisonous snakes, do not show evidence of poisoning, even though the fangs have penetrated the skin, "dry bites".

Snake Venom: The salivary glands of the snake are situated behind the eyes. Venom is the saliva of the snake. Cobra venom is faint-transparent, yellow and is slightly viscous. Russell's viper venom is white or yellow. Venom travels in the body through lymphatics and superficial veins. Snake venoms are complex mixtures, chiefly proteins, many of which have enzymatic activities. Enzymes include phospholipidase A, proteases, hyaluronidase, cholinesterase, hydrolases, transaminase, phospholipase, ATPase, ribonuclease, deoxyribonuclease, etc.

Action: The colubrine venom is mainly neurotoxic and has a primary toxicity for the respiratory and cardiac centres. It can produce marked cardiac or vascular changes, or have a direct effect on the blood. The viperine venom is mainly haemolytic and causes intravascular haemolysis and depression of the coagulation mechanism. It can also produce changes in the nervous system or in vascular dynamics. As a rule one of the modes of action far exceeds the other. The sea snake venom is myotoxic.

Table (28-1). Difference between Cobra and Viper

Trait	Cobra	Viper
(1) Body:	Long and cylindrical.	Short; narrow neck.
(2) Head:	Small; seldom broader than body; covered by large scales or shields of special forms.	Large; broader than body; triangular and covered by numerous small scales.
(3) Pupil:	Round.	Vertical.
(4) Maxillary bone:	Carries poison fangs and other teeth.	Carries only poison fangs.
(5) Fangs:	Grooved, short, fine.	Canalized, long.
(6) Venom:	Neurotoxic.	Hemotoxic.
(7) Tail:	Round.	Tapering.
(8) Eggs:	Lay eggs.	Give birth to young ones.

Table (28-2). Difference between poisonous and non-poisonous snakes

Trait	Poisonous	Non-poisonous
(1) Head scales:	(1) Small.	Large
(2) Belly scales:	Large and cover entire breadth.	Small, like those on the back or moderately large, but do not cover the entire breadth.
(3) Fangs:	Hollow like hypodermic needles.	Short and solid.
(4) Teeth:	Two long fangs.	Several small teeth.
(5) Tail:	Compressed.	Not much compressed.
(6) Habits:	Usually nocturnal.	Not so.

Signs and Symptoms: Ophitoxaemia is **poisoning by snake venom. The most common symptom following snake bite (poisonous or non-poisonous) is fright, especially the fear of rapid and unpleasant death.** Due to fright, the victim may become semiconscious with cold clammy skin, feeble pulse and rapid breathing. These emotional symptoms appear within few minutes of the bite. **Sometimes, it produces psychological shock and even death.** It may also give rise to tetanus or gas gangrene. A viperid snake merely strikes, discharging the venom, the moment the fangs penetrate the skin and then immediately leaves it. In cobra bite, the transfer of venom is not complete.

Cobra bite: Local symptoms start within 6 to 8 minutes. **A small reddish wheal develops at the site of bite.** The bitten area is tender with slight radiating burning pain and **oozing of blood-stained fluid**. Swelling may be minimal or even absent. **Systemic symptoms appear after about half hour.** The patient feels sleepy, slightly intoxicated, weakness of legs, and is unable to stand or move. Nausea and vomiting are sometimes the early symptoms. **Ptosis is the earliest neuroparalytic manifestation followed by ophthalmoplegia.** There may be extraocular muscle weakness and strabismus. **Weakness of the muscles increases, and develops into paralysis of the lower limbs.** After half to one hour, there is excessive salivation and even vomiting, headache, vertigo, paraesthesia around the mouth and myalgia. This is followed by **paralysis and swelling of the tongue and the larynx, due to which there is difficulty in speech and swallowing. The facial muscles, palate, jaws, tongue, vocal cords, neck muscles and muscles of deglutition become progressively flaccidly paralysed. After about two hours, the paralysis is complete. Respiratory arrest may occur** due to obstruction of upper airway by the paralysed tongue or inhaled vomitus, or due to paralysis of intercostal muscles and diaphragm. Respirations become slower and the heart rate increases. **Though the patient is conscious, he is not able to speak.** Coma sets in and finally the respiration stops with or without convulsions and the heart stops.

Krait bite: Symptoms resemble those of cobra bite, but there is no swelling or burning pain at the site of the bite and the convulsions are milder, while the feeling of drowsiness and intoxication is more intense. Albumin appears in urine.

Russell's Viper and Echis Carinate bite: More than 50% of the victims have minimal or no poisoning, as little or no venom is injected. About 25% will develop serious generalised poisoning but death occurs rarely. When venom is injected, **the spot develops severe pain within 8 minutes.** The area round the bite is red and painful. The onset of swelling starts within 15 minutes and there is often blood-stained discharge from the wound. **Persistent bleeding from bite site is a constant feature. Blisters begin to appear in about 12 hours** in and around the bite site, progressing subsequently to involve the entire limb. When the amount of venom injected is less, pain and swelling restricted to below the elbow or knee and nausea disappear within one to two days. In moderate poisoning, there is a marked feeling of intense pain, vomiting, giddiness, sweating, abdominal pain, dilatation of the pupils getting insensitive to light, and in about one to two hours there is marked collapse and often complete loss of consciousness. Skin temperature is

Table (28-3). Difference between Colubrine and Viperine bites

Trait	Colubrine bite	Viperine bite
(1) Area bitten:	Reddish wheal; tender with burning pain; oozing of blood-stained fluid less.	Pain and oozing of blood-stained fluid are more
(2) Swelling:	Minimal or absent in bitten area.	Involves limb and spread up to trunk
(3) Symptoms:	Neurotoxic.	Haemotoxic
(4) Speech and deglutition:	Difficult.	Normal.
(5) Paralysis:	Of lower limbs, spreading to trunk and head	Not present.
(6) Salivation:	Present.	Absent.
(7) Pupils:	Normal.	Dilated, do not react to light.
(8) Blood pressure:	Normal.	Hypotension.
(9) Bleeding and coagulation time:	Normal.	Prolonged.
(10) Haemorrhagic manifestations:	Not present.	Prominent feature.
(11) Cause of death:	Respiratory failure.	Shock and haemorrhage.

raised. **Tingling and numbness over the tongue and mouth** or scalp and paraesthesia around the wound occur. **These symptoms usually subside within few hours. There is local extravasation of blood and swelling spreads as far as the trunk in one to two days, without further generalised symptoms.** Though the limb is swollen and red, it is usually not ~~tender~~. **Haematuria** may be seen within a few hours of the bite. **In severe cases, the main feature is the persisting shock. A haemorrhagic syndrome with epistaxis, haemoptysis, ecchymoses, intracranial and subconjunctival haemorrhages, and bleeding into the floor of the mouth, tympanic membrane, G.I. and G.U. tract, retroperitoneum and intraperitoneum, etc., occur due to the increased coagulation time**. Urine contains blood, sugar and protein. Intravascular haemolysis may lead to haemoglobinuria and renal failure. **Petechial haemorrhages are common. The prothrombin time is markedly increased. In systemic poisoning, the blood becomes defibrinated and therefore will not clot.** Increasing respiratory depression, blurring of vision, headache, dizziness and weakness often occur. Towards the end, there is an extensive suppuration and sloughing, followed by a malignant oedema of the bitten area. Paralysis does not occur. Death is usually due to shock and haemorrhage. In the case of *Echis*, death may not occur, but the secondary symptoms continue for days and the haemorrhages are severe, and the wound shows mild necrosis (Table 28-3).

Sea Snakes bite: Symptoms are not produced in about 80% of bites. Bites cause little or no local reaction. After half to one hour, the patient develops pain, stiffness and weakness of the skeletal muscles. **Sea snake bites result in marked polymyositis with a limb-girdle distribution.** Trismus occurs in early stage. Later, flaccid paralysis develops, beginning with ptosis. Myoglobinuria with renal failure may occur. Marked weakness of muscles persists for several months. Death may occur due to cardiac arrest or paralysis of respiratory muscles.

Fatal Dose: Cobra: 12 mg; Russell's viper: 15 mg; echis: 8 mg; krait: 6 mg; of dried venom. The average yield in one bite in terms of dry weight of lyophilised venom is: Cobra: 170 to 325 mg; Russell's viper: 130 to 250 mg; krait: 20 mg; and echis: 20–35 mg.

Fatal Period: Cobra half to six hours; viper one to four days.

Diagnosis: (1) **Snake specific venom antigens** have been detected in wound swabs, aspirates or biopsies, serum, CSF, urine and other body fluids. As such skin and underlying tissues surrounding

the fang punctures, wound and blister aspirates and urine should be collected. Urinary venom may remain detectable even though the victim is treated with anti-venom. (2) **Radioimmunoassay (RIA)** is most sensitive and specific which can detect venom levels of 0.4 µg/l. Bitten area of skin should be preserved. (3) **Enzyme immunoassay (EIA)** is commonly used as it is simple and can detect venom levels of 5 µg/l. (4) Immunological detection of venom antigens in body fluids can be done by **ELISA**. (5) A swab taken from the wound site or extract from the skin is **injected into a frog** for evidence of toxicity.

First Aid: (1) **Assure the patient.** (2) Immediately **apply a broad firm bandage** (Sutherland wrap) around the limb and on the bitten area for elapid and sea snake bites, but not for viper bites as it may cause local necrosis. As much of the limb should be bandaged as is possible. It should be tight enough (50 to 70 mm Hg) to occlude the superficial venous and lymphatic return, but not the arterial or deep venous flow. In bites on the trunk, head or neck, apply firm pressure over the bitten area. (3) **Immobilise the limb,** as movement can accelerate the spread of venom. Avoid elevation of an extremity, as it may hasten systemic absorption of venom. (4) Local incision and suction should not be done as it causes local bleeding and nerve injury. (5) Do not suck venom out of the wound, and do not use chemicals or medicines on the wound. (6) Cryotherapy is not of much use. (7) The **wound should not be cauterised** as it actually seals the poison within the tissues. (8) Clean the wound with sterile saline or water, and cover with a sterile dressing. (9) Make the patient lie on one side in the recovery position, so that the airway is clear, in case of vomiting or fainting.

If a patient is brought after a few hours (4 to 6) of snake bite with mild local swelling and no systemic symptoms, he should be kept under observation for 24 hours, closely monitored for vital signs, cardiac system and oxygen saturation; given tetanus toxoid and discharged.

Treatment: (1) **Polyvalent anti-snake venom (PAV)** is prepared by hyperimmunising horses against the venom of the four common poisonous snakes, i.e., cobra, common krait, Russell's viper and saw-scaled viper. Plasma obtained from the hyperimmunised horses is concentrated and purified. The serum is lyophilised by drying it from the frozen state under high vacuum. It is prepared in the Haffkine Institute, Mumbai, King Institute, Chennai, Serum Institute, Pune, and at Kasauli in India, and is available in the form of lyophilised powder in an ampoule, which retains potency for about five years. **It is useful when given within 4 hours, and is of doubtful value after 24 hours.** Each vial of PAV will neutralise about 6 to 8 mg of venom. Its half-life is about 90 hours. Test dose of PAV should not be administered as it is a poor predictor of early anaphylactoid reactions and may presensitise the patient to PAV. **If the swelling involves at least half of the bitten limb within a few hours of bite in the absence of a broad firm bandage, or if the swelling rapidly continues or if swelling has crossed a joint within first one or two hours of its starting, it indicates envenomation. In case of viper bites, incoagulable blood or evidence of spontaneous bleeding from the gums will indicate envenomation.**

Dose: (1) **Minimal symptoms:** Local swelling but no systemic reactions, 5 vials. (2) **Moderate:** Swelling progressing beyond site of bite with systemic reaction, 10 vials. (3) **Severe:** Marked local reaction, severe symptoms, 10 to 15 vials. The lyophilised powder is diluted in 500 ml of distilled water or normal saline and infused over a period of one hour. In neurotoxic poisoning, a second dose of ten vials should be given after one hour. In the case of haemotoxic patients, the initial dose of antiserum will be neutralising unbound, free flowing venom. **Children require the same dose as in adults.** The liver requires six hours to restore clotting factors, as such patient will not require further PAV for 6 hours after the first dose.

20-minute blood clotting test: Few millilitres of fresh venous blood is put in a clean dry glass tube and left undisturbed for 20 minutes and then gently tilted. If the blood is still liquid it indicates viper bite. This test is repeated every 6 hours to determine repeat dose requirement. Normalisation of clotting time is taken as endpoint of therapy.

Other measures: At the first sign of any of the following, e.g. urticaria, itching, shivering, chills, nausea or vomiting, hypotension, bronchospasm, angiooedema: (1) (A) Stop PAV infusion. (B) Administer 0.5 mg. 1 : 1000 adrenaline I.M. for adults

(0.01 mg/kg for children). (C) Give hydrocortisone and antihistamine to provide longer term protection. (D) If there is no improvement after 10 to 15 minutes give a second dose of adrenaline. (E) Once the condition has improved start antiserum infusion. (2) **If the anti-snake venom is not available, 40 ml. of antivenin is given i.v. and repeated as required. It is effective for cobra and Russell's viper bites.** When treating viper bite, a watch should be kept on prothrombin time. (3) If there are signs of neuroparalysis, give 1.5 mg neostigmine for adults (paediatric dose 0.04 mg/kg) I.M. with 0.6 mg atropine I.V. (paediatric dose 0.05 mg/kg) to counteract the muscarinic effects of neostigmine. It is repeated twice at 10 minutes interval. If the victim shows improvement, give 0.5 mg neostigmine half hourly with atropine. In cases of presynaptic envenoming, such as kraits or Russell's viper a positive response does not occur. (4) Before every injection half mg. atropine should be given to block muscarinic side effects. (5) Heparin thousand to five thousand i.u. may be given i.v. if there are clotting abnormalities. (6) Inject tetanus antitoxin. (7) A broadspectrum antibiotic should be given if there is severe tissue involvement. (8) In viper poisoning, sedatives may be given to relieve pain and nervousness. (9) In case of collapse, general stimulants are of value. (10) In severe poisoning, infusion of normal saline or transfusion of blood or plasma is very useful. (11) **Haemodialysis** may be necessary. Peritoneal dialysis is better. (12) Give paracetamol for pain, but aspirin should not be given, as it may make the patient bleed. (13) Surgical debridement of the blebs, bloody vesicles, and superficial necrosis may be necessary.

Postmortem Appearances: Poisonous snakes leave two or occasionally one fang mark. Non-poisonous snakes leave a semicircular set of tooth marks. The punctures are one and one-fourth cm. deep in colubrine and 2.5 cm. deep in viperine bites. Sometimes, the bite marks may not be visible. **In colubrine bite, the site of bite contains fluid and haemolysed blood causing staining of vessels. Haemorrhages** into the bowel, purpuric spots on pericardium and haemorrhages in the lungs and in many tissues may be seen. Kidneys are inflamed. Internal organs are congested. Washing from the bite area may contain cholinesterase or thromboplastin.

Absorption and Excretion: Snake venom is poisonous only when injected, and is harmless when taken by the mouth, as it will be digested by enzymes in intestinal tract and absorbed as amino acids. It is excreted by kidneys, milk and probably by salivary glands and the mucous membranes.

Poisoning: Poisoning is always accidental. Occasionally, a murder is committed by throwing a poisonous snake on the bed of sleeping person. Cattle are sometimes poisoned by snake venom. For this, a cobra is shut up in an earthen vessel containing a banana, and heat is applied to the vessel. The snake being irritated, bites the fruit and the venom is injected to the pulp, which is crushed and smeared on a rag. The rag is thrust into the animal's rectum by means of a split bamboo. **The bodies of animals killed by snake poisoning may be eaten without ill-effects, but their blood is poisonous and is fatal if injected into the human body.**

SCORPIONS

These are eight-legged arthropods and have a hollow sting in the last joint of their tail. The venom is a clear, colourless toxalbumen and can be classified as either haemolytic or neurotoxic. Its toxicity is more than that of the snakes, but only a small quantity is injected. The mortality, except in children is negligible. There are more than 1250 species of scorpions. Colour of scorpions varies from light yellow to black (Plate 20-120).

SIGNS AND SYMPTOMS: If the scorpion has haemolytic venom, the reaction is mainly local and simulates the viper snake bite, but the scorpion sting will have only one hole in the centre of the reddened area. The extremity will have oedema, pain and reddening. This usually lasts for one to two hours. The symptoms produced by neurotoxic venom are similar to cobra bite.

TREATMENT: A tourniquet should be applied above the location of sting. Pack sting in ice, and incise and use suction, and wash the wound with a weak solution of ammonia, borax or potassium permanganate. A local anaesthetic (2% novocaine or 5% cocaine) is injected at the site to lessen pain. A specific antivenin is available for most species. Calcium gluconate intravenously is of value to control local swelling. Barbiturates should be given to reduce excitement and convulsions but morphine is contraindicated.

BEES AND WASPS: The venom of bees, wasps, hornets, and ants is a complex mixture of biochemical compounds ranging from simple amines to complicated proteins and enzymes. Painful and sometimes fatal reactions occur in humans. The reactions are usually

local. Severe systemic reactions are not common. Venom contains histamine. Stings of the mouth, throat and sometimes of the face, neck or limbs cause oedema of the larynx or pharynx and obstruction. Systemic toxic reactions occur due to multiple stings. There are gastrointestinal disturbances and shock. Vomiting and diarrhoea may be accompanied by faintness and unconsciousness. The symptoms last for one day, if not fatal.

The anaphylactic reaction may occur immediately or within 20 minutes. There is respiratory distress, faintness, and unconsciousness. A rash may develop. Death may occur in 2 to 15 minutes.

TREATMENT: (1) Apply a ligature above the site of the sting. (2) The sting should be located and removed. (3) Tincture of iodine or local application of antihistamine is useful. (4) Adrenaline is given to combat systemic reactions. (5) ACTH 25 mg. in a litre of normal saline is given by intravenous infusion. (6) Calcium intravenously is useful for urticaria and oedema.

CENTIPEDES: They have segmented bodies with a pair of legs on each segment and a pair of claws on the first segment, through which venom is injected. The length is from two to several centimetres. The colour may be greenish-black or black (Plate 22-133). Toxins are histamine, serotonin, hyaluronidase, esterase and proteinases.

SYMPTOMS: Local swelling, excruciating pain, necrosis, paralysis and contracture of extremities, cardiac irregularities, arthritis, regional lymphadenopathy, and meningism may occur. Symptoms subside in 2 to 3 days.

LIZARDS (Plate 22-132): There are no venomous lizards in India.

ANTS: They secrete formic acid by certain glands situated in the tail. Ant bite produces pain, irritation and swelling at the site of bite.

SPIDERS: The venoms are either cytotoxic or neurotoxic, and act on the myoneural junction or peripheral nerve endings causing ascending motor paralysis or damage to peripheral nerve endings. Spider fangs leave two puncture marks on the skin. Local swelling and local muscle spasms appear, which later involve muscles of the back, shoulder, thighs, legs, arms and face. There is circulatory collapse, convulsions and delirium. It may be confused with tetanus and strychnine poisoning. Death may occur due to introduced infection rather than venom. Antivenin and adrenaline are given.

CHAPTER

29 CNS Depressants

Classification: (1) Ethyl alcohol. (2) General anaesthetics. (3) Opioid analgesics. (4) Sedatives and hypnotics.

Inebriant poisons produce intoxication, i.e., lightheadedness, confusion, disorientation and drowsiness, e.g. alcohol, barbiturates, chloral hydrate, benzodiazepines, paraldehyde, anaesthetics, hydrocarbons, etc.

ALCOHOL

The term alcohol in common use refers to ethyl alcohol. It is a transparent, colourless, volatile liquid, having a characteristic spirituous odour and a burning taste. Absolute alcohol contains 99.95% alcohol; rectified spirit contains 90% alcohol. **"Proof spirit"** is one **which at 10.5°C weighs exactly 12/13 part of an equal measure of distilled water.** Weaker spirits are termed "underproof" and stronger spirits "overproof". Ethanol labelled 100 proof is 50% ethanol.

Concentration: The approximate percentage of alcohol by volume in some beverages is as follows:

Vodka	60 to 65%
Rum and liquors	50 to 60%
Whisky, gin, brandy	40 to 45%
Port, sherry	20%
Wine	10 to 15%
Beers	4 to 8%

Arrack: It is a liquor distilled from palm, rice, sugar or jaggery, etc. and has a strength of 40 to 50%. It may be mixed with chloral hydrate and potassium bromide for getting a greater kick.

ABSORPTION: Absorption from the stomach and small intestine begins almost immediately upon ingestion. About 20% is absorbed from the stomach and 80% through small intestine. Alcohol can be detected in the blood within 2 to 3 minutes of swallowing. The maximum concentration in blood is reached within 45 to 90 minutes after ingestion. Ethanol is distributed evenly throughout the body water, passing the blood-brain barrier easily. It is poorly soluble in body fat and as such females of the same body weight, will have a higher (25% higher) blood alcohol concentration for the same amount of drink, as their aqueous compartment is smaller. About 60% of alcohol inhaled can be absorbed into systemic circulation.

METABOLISM: About 90% of alcohol absorbed is oxidised in the liver, and the remaining 10% is excreted mainly by the kidneys and the lungs. In the liver, alcohol is oxidised to acetaldehyde by alcohol dehydrogenase (ADH) and its coenzymes, nicotinamide adeninedinucleotide (NAD). In the second step, acetaldehyde is transformed into free acetic acid or its activated form, acetyl coenzyme A. Finally, the acetate enters the general pool and undergoes oxidation to CO_2 and water in the citric acid (Krebs) cycle. During its oxidation alcohol is not stored in the tissues. It disappears from the blood at a fairly uniform rate of about 10 to 15 ml. per hour. This is the equivalent of about 15 mg.% from the blood per hour. 10% of metabolised alcohol is deposited in the tissues as lipids in the form of cholesterol and neutral fat.

Action: Alcohol is a well-known stimulant, but is a selective depressant, especially of the higher nervous centres which it inhibits. It is not a true aphrodisiac. It is a hypnotic and diaphoretic.

Symptoms: (1) Stage of Excitement: There is first a feeling of well-being and a certain slight excitation. The actions, speech and emotions are less restrained. **There is increased confidence and a lack of self-control**, which is a constant feature of alcoholic poisoning. The person may disclose secrets. Normal good manners are forgotten. The neat and orderly are careless in their dress. There may be increase of sexual desire. **Strong light is often needed to distinguish objects and dimly lighted objects may not be distinguished at all.** It takes longer to see clearly again after being dazzled by a strong light. **When jerking movement is in the direction of the gaze and independent of the position of the head, it is known as alcohol gaze nystagmus**, and **appears at blood levels of 40 to 100 mg.% (average 80 mg.%)**. It is not a constant or common sign. **Mental concentration is poor and judgement impaired.** These effects are usual between 50 to 150 mg./100 ml. of blood alcohol.

(2) Stage of Incoordination: Between 150 and 250 mg/100 ml., the sense perceptions and skilled movements are affected. **Alteration in the conduct of the individual occurs.** He may become carefree, cheerful, ill-tempered, irritable, excitable, quarrelsome, sleepy and so on, according to the dominant impulses which have been released. **There is certain clumsiness and incoordination in the fine and more skilled movements** as shown by slight alteration in speech and in the fine finger movements. Nausea and vomiting are common. The breath smells of alcohol. The face is flushed and the pulse rate is increased. Sense of touch, taste, smell and hearing are diminished. The pupils are dilated. The temperature becomes subnormal.

(3) Stage of Coma: In this stage, the motor and sensory cells are deeply affected, speech becomes thick and slurring, coordination is markedly affected, causing the patient to become giddy, stagger and possibly to fall. The person passes into a state of coma with stertorous breathing. The pulse is rapid and temperature subnormal. **The pupils are contracted, but stimulation of the person, e.g., by pinching or slapping, causes them to dilate with slow return (Mc Ewan sign). This sign occurs at or above 300 mg.% of blood level of alcohol.** Death occurs from asphyxia due to respiratory paralysis but it may occur from shock (**Table 29-1**).

CAGE questionnaire is an internationally used assessment instrument for identifying alcoholics.

MICTURITION SYNCOPE is a condition which occurs usually after heavy beer drinking. When the person rises from the bed in the middle of night to pass urine, he loses consciousness during the act of urination, probably due to sudden upright posture.

Munich Beer heart is a condition in which cardiac dilatation and hypertrophy is seen due to excessive and prolonged beer drinking.

Fatal Dose: 150 to 200 ml. of absolute alcohol consumed in one hour.

Fatal Period: 12 to 24 hours.

Tolerance to Alcohol: It is acquired and may be lost by those 'out of practice'. It is restricted by liver damage. A person in the habit of taking alcohol daily can drink alcohol without getting drunk in quantities which would seriously affect a person unaccustomed to taking it.

Treatment: (1) Evacuation of the stomach and bowel. (2) The patient must be kept warm. (3) Fructose 300 g. by infusion over half-hour increases the rate of fall of alcohol by about 25% by promoting its metabolism. (4) One litre of normal saline with 10% glucose, 100 mg. thiamine, and 15 units of insulin are useful. (5) Artificial respiration if there is difficulty in breathing. (6) Inhalation of oxygen is of great value. (7) Haemodialysis or peritoneal dialysis is very useful.

Postmortem Appearances: On opening the cavities of the body, alcoholic odour is frequently noted. Acute inflammation of stomach with a coating of mucus is found. The brain, liver and lungs are congested and the smell of alcohol in the viscera may be noted. The blood is usually fluid and dark. Oedema and congestion of the brain and meninges are seen. **In embalmed bodies alcohol is estimated on either vitreous or muscle.**

Table (29-1). Physiological effects of alcohol

Blood alcohol concentration	Effects
0 to 50 mg%	No significant effect or mild euphoria.
50 to 100 mg%	Decreased inhibitions, increased self-confidence, decreased attention span, alteration of judgement, nystagmus.
100 to 150 mg%	Some mental confusion, emotional instability, loss of critical judgement, impaired memory, sleepiness, slowed reaction time.
150 to 300 mg%	Loss of muscular coordination, staggering gait, marked mental confusion, exaggeration of emotions, dizziness, decreased pain response, disorientation, thickened speech.
300 to 400 mg%	Stupor, marked incoordination, marked decrease in responses to stimuli, possibly coma.
400 mg% and above	Anaesthesia, depression of responses, deep coma, death.

Chronic Poisoning: Alcohol addicts are people who cannot stop drinking for long, or who experience withdrawal symptoms, if they do. **Chronic alcoholics** are those, who have reached a state of irreversible somatic or brain changes caused by alcohol. The patient suffers from nausea, vomiting, anorexia, diarrhoea, jaundice, tremors of the tongue and hands, insomnia, loss of memory, impaired power of judgement, hypoproteinaemia and general anasarca. The symptoms of peripheral neuritis and dementia supervene in the last stage. Such patients generally die suddenly from coma.

Postmortem Appearances: The gastric mucous membrane is deep reddish-brown, with patches of congestion or effusion and is hypertrophied. The liver is congested and shows fatty infiltration, enlarged or cirrhotic or contracted. The kidneys show granular degeneration. The heart is dilated and shows fatty degeneration.

Treatment: (1) **Antabuse** (disulfiram) is given in a single daily dose of 250 mg. It inhibits aldehyde dehydrogenase. The dosage is gradually reduced until an adequate daily dosage of 125 to 250 mg. is reached, which should be continued until the patient has been conditioned to accept adequate follow-up therapy. Antabuse leads to accumulation of acetaldehyde in the blood and tissues and causes unpleasant symptoms, due to which the patient dislikes alcohol. (2) **Citrated calcium carbimide** (temposil) 50 mg. once a day can be used with less side-effects. (3) Chlorpromazine 25 to 50 mg. every 4 to 6 hours is also useful. (4). Clonidine 60 to 180 mg/hr i.v. (5) Chlormethiazole.

(4) The Conditioned Reflex Treatment: It consists of giving alcoholic beverages to the patient in surroundings that affect his visual and olfactory senses. With a backdrop of bottles of various alcoholic beverages, the patient is given various types of liquor, together with drugs that will cause immediate and acute nausea and vomiting. After 5 to 8 daily treatments, symptoms are brought on simply by the sight of a bottle, and the patient begins mentally to associate his painful sickness with the alcohol. Hypnosis and psychotherapy are also useful.

Alcoholics Anonymous is an international organisation, which has branches throughout India. It consists mainly of ex-alcoholics who usually meet once a week, share their experiences and try to encourage one another to abstain from alcohol.

DRUNKENNESS

Drunkenness is a condition produced in a person, who has taken alcohol in a quantity sufficient to cause him to lose control of his faculties to such an extent, that he is unable to execute safely the occupation in which he is engaged at the particular time.

The clinical diagnosis depends on the combination of a number of symptoms and signs, no single one of them being peculiar to this condition, except the odour of alcohol from the breath. An individual can react differently under different circumstances, and that the same amount of alcohol can produce different effects on different people under same circumstances.

A Model Scheme of Medical Examination: (1) Exclusion of Injuries and pathological states: The following conditions which simulate alcoholic intoxication should be excluded: (a) Severe head injuries. (b) Metabolic disorders, e.g., hypoglycaemia, diabetic precoma, uraemia, hyperthyroidism. (c) Neurological conditions, e.g., disseminated sclerosis, intracranial tumours, Parkinson's disease, epilepsy, acute aural vertigo. (d) Drugs: insulin, barbiturates, antihistamines, morphine, atropine. (e) Certain pre-existing psychological disorders, e.g., hypomania, general paresis. (f) High fever. (g) Exposure to CO.

(2) History: The history of the relevant events should be obtained from the accused person while observing him. Enquire whether he suffers from any disease or disability and whether he is under medical treatment.

(3) General behaviour: (a) General manners and behaviour. (b) State of dress: Presence of slobber on mouth or clothing; presence, character and colour of any vomit, soiling of clothes by excretions. **(c) Speech:** Note the type, e.g., is it thick, slurred or overprecise? Slight blurring of certain consonants is one of the earliest signs of incoordination of the muscles of the tongue and lips. Certain test phrases may be used to bring out this difficulty in speech, such as "British Constitution", "West Register Street", "Truly Rural", etc. A sober individual will say that he is not good at such phrases; the semi-intoxicated person will often insist on getting them correctly. **(d) Self-control:**

Whether he is able to control himself in response to the demands made on him by the examiner.

(4) Memory and mental alertness: The memory of the person for recent events, and his appreciation of time can be judged by asking suitable questions about his movements during the preceding few hours, and the details of his accident if any. A few very simple sums of addition or subtraction may be asked.

(5) Handwriting: The examinee should be asked to copy a few lines from a newspaper or book. A note should be made of: (a) The time taken. (b) Repetition or omission of words, letters, or lines. (c) Ability to read his own writing. The examinee should be asked to sign his name. The signature can be compared with that on his driving license if any.

(6) Pulse: The resting pulse should be taken at the beginning and at the end of the examination. The pulse is rapid and is usually full and bounding.

(7) Temperature: The surface temperature is usually raised.

(8) Skin: Skin is warm, dry and flushed.

(9) Mouth: (a) Record the general state of mouth, teeth and tongue, noting whether the tongue is dry, furred or bitten. (b) The smell of the breath should be recorded.

(10) Eyes: (a) **General appearances:** (i) Whether the lids are swollen or red, and whether the conjunctivae are congested. (ii) The colour of the eyes, and abnormalities. (b) **Visual acuity:** Any gross defect should be noted. (c) **Intrinsic muscles:** (i) **Pupils:** Equal or unequal, dilated or contracted or abnormal in any way, (usually dilated in early stages, but may be contracted in later stages or coma). (ii) **Reaction to light:** Note whether the action is brisk, slow or absent. They may become unequal, equalising again in response to light, and dilate again slowly even if the light continues to be directed into the eyes. (d) **Extrinsic muscles:** (i) **Convergence:** Test the degree of ability to follow a finger in all normal directions and to converge the eyes normally on a near object. (ii) **Strabismus:** Note whether it is present. (iii) **Nystagmus:** The presence of fine lateral nystagmus may indicate alcoholic intoxication.

(11) Ears: Examine for (a) gross impairment of hearing, (b) abnormality of the drums.

(12) Gait: The examinee should be asked to walk across the room and note: (a) **Manner of walking:** Is it straight, irregular, overprecise, unsteady, reeling, or with feet wide apart? (b) **Reaction time to a direction to turn:** Does the examinee turn at once or continue for one or two steps before obeying? (c) **Manner of turning:** Does the examinee keep his balance, lean forward, or swing to one side? Does he correct any mistake in a normal or exaggerated way? It is undesirable to ask examinee to walk along a straight line drawn on the floor.

(13) Stance: Note whether the examinee can stand with his eyes closed and heels together without swaying (Romberg's sign).

(14) Muscular coordination: Ask the examinee to perform the following tests. (a) Placing finger to nose. (b) Placing finger to finger. (c) Picking up medium-sized objects from the floor. (d) Lighting a cigarette with a match. (e) Unbuttoning and rebuttoning coat.

(15) Reflexes: Knee and ankle reflexes should be tested which are delayed or sluggish. Plantar reflex may be extensor or flexor.

(16) Pulmonary, cardiovascular and alimentary systems: The heart, lungs and abdomen should be examined and the blood pressure taken to establish the presence or absence of disease.

(17) Laboratory Investigations: The degree of intoxication can be estimated by the concentration of alcohol in the blood, urine, breath and saliva.

URINE: The disadvantages of urine examination are: (1) A time lag before equilibrium between blood and urine is reached; the maximum concentration is reached about 20 to 25 minutes later than in blood. (2) The urine alcohol concentration at any given time after the maximum concentration in blood has been reached will be higher by 20 to 30% than in the blood, because the specimen of urine examined has been secreted from the blood at some earlier period. (3) Alcohol may pass through the lining of the bladder in either direction both in life and after death, depending on the relative concentration of alcohol in blood and urine.

Analyses of two urine samples are required. The first sample should be taken as soon as possible following the incident, the bladder being completely emptied. The second sample should be taken half-an-hour later. The concentration of alcohol in the second specimen reflects the blood alcohol level during the interspecimen interval.

50 ml of phenyl mercuric nitrate or sodium fluoride for 10 ml. are used as a preservative for urine which causes bacterial inhibition.

Collection of Blood: Spirit must not be used for cleaning the skin. The skin is cleaned with a solution of 1:1000 mercuric chloride or washed with soap and water. **Blood samples should be preserved by the addition of 100 mg. of sodium fluoride and 30 mg. of potassium oxalate for 10 ml. followed by thorough shaking. This prevents loss of alcohol by glycolysis and bacterial action.** A screw-capped glass bottle of "universal" size is suitable. The container should be tightly clamped and sealed to prevent loss of alcohol by evaporation. Blood is the most suitable and the most direct evidence of the concentration of alcohol in the brain. **The disadvantages are:** (1) It may be difficult to collect from an uncooperative person. (2) Consent of the person is necessary. (3) Substances like acetone, ether, paraldehyde, etc., when present in the blood are estimated as alcohol. When blood is kept in a refrigerator, formation of oxidisable substances is not significant. **Blood samples should not be frozen, as freezing will cause the cells to lyse.**

Widmark evolved a formula which takes into consideration the size and sex of the subjects, and the type of alcoholic liquor consumed. The formula is: a = prc, where a, is weight of alcohol (in g.) in the body, p, is the body weight (in kg.), c, is the concentration of alcohol in the blood (in mg. kg.), and r is a constant (0.68 for men and 0.55 for women). For urine analysis, the formula is a = 3/4 prq. q is the alcohol concentration (in mg. kg.).

Breath: About 2100 ml. of alveolar air contains the same amount of alcohol as one ml. of blood. The temperature of breathed out air is 34°C. The person is asked to blow into the apparatus. Sophisticated versions based on fuel-cell sensing, electrochemical oxidation, and microprocessors have come into use. They are a direct method which instantly measure breath alcohol quantitatively and provide a printout.

Saliva and vitreous can also be examined.

Diagnosis of Drunkenness: The usual signs of drunkenness are: strong odour of alcohol in breath, loss of self-control and loss of clearness of intellect, unsteady gait, vacant look, congested eyes, sluggish and dilated pupils, dry lips, increased pulse rate, unsteady and thick voice, talks at random and lack of perception of passage of time.

The effects of acute alcohol intoxication are more marked when the blood level is rising than falling. The individual's psychological reaction to acute alcohol intoxication is variable. **The medical examination is extremely subjective; many of the observations made are incapable of objective or quantitative record.** There is individual variation from one clinical examiner to another. If an arrested person suffers from fear or acute anxiety often complicated by fatigue states, the signs may simulate drunkenness. A chronic alcoholic with a blood level of 150 mg% may appear sober though there is impairment in the reflexes, visual acuity, memory, concentration, and judgement.

Determination of blood alcohol helps: (1) to know the concentration of alcohol circulating in the body, (2) to determine within fairly wide limits how much alcohol must have been imbibed within a certain period of time, which can help the Court in testing the reliability of the statements of the accused, (3) to assist the doctor in confirming his suspicions, (4) enable the Court to accept clinical diagnosis, (5) serve to resolve conflicting reports given by non-medical witnesses.

Medical Terminology: "Under the influence" means that due to drinking alcohol, a person has lost some of the clearness of the mind and self-control that he normally possesses. All individuals with a blood alcohol level of 140 mg% are intoxicated to the point where they cannot deal with unusual, emergency or non-customary problems.

Below 10 mg%	:	Sober.
10 to 70 mg%	:	Drinking.
80 to 150 mg%	:	Under the influence.
150 to 300 mg%	:	Drunk or intoxicated.
300 to 400 mg%	:	Stupor.
400 mg% and above	:	Coma, and death.

In some countries the law has made it an offence for a person to drive a motor vehicle above a specified blood alcohol level, e.g., 20 mg% in Sweden, 80 mg% in U.K., and France, and 100 to 150 mg% in different States of USA. **The statutory limit in India is 30 mg% (S. 185, Motor Vehicle Act, 1988).**

Pathology: (1) Delirium Tremens: This results from the long continued action of the poison on the brain. It occurs in chronic alcoholics due to: (1) temporary excess, (2) sudden withdrawal of

alcohol, (3) shock after receiving an injury, such as fracture of a bone, or (4) from acute infection, such as pneumonia, influenza, erysipelas, etc.

It typically begins 72 to 96 hours after the last drink. There is an acute attack of insanity in which the main symptoms are coarse muscular tremors of face, tongue and hands, insomnia, restlessness, loss of memory, psychomotor uncontrollable fear and tendency to commit suicide, homicide or violent assault or to cause damage to property. **There is disorientation as to time and place, and a peculiar kind of delirium of horrors owing to hallucinations of sight and hearing. The patient imagines that insects are crawling under the skin, or snakes are crawling on his bed. It is considered unsoundness of mind, and not intoxication.**

(2) Alcoholic polyneuritis.

(3) Alcoholic paranoia.

(4) Acute alcoholic hallucinosis.

(5) Alcoholic epilepsy: Seizures occur after a day or more of the termination of a drinking session. Sometimes, the attacks may occur while the patient is actually drinking.

(6) WERNICKE'S ENCEPHALOPATHY: This results from a brain or spinal cord lesion due to heavy drinking. Symptoms include disturbance of consciousness (ranging from mild confusion to coma), ophthalmoplegia, nystagmus, clouding of consciousness, ataxia, delirium and stupor. It has a high mortality and can cause death in 24 hours. If untreated it can progress to a more chronic condition known as Korsakoff psychosis. The main features are psychotic personality, polyneuritis, insomnia, wasting, unsteady gait, illusions, hallucinations and amnesia and confabulation.

Hazards of Alcohol: Fatal acute poisoning by alcohol is rare, but mild and moderate degrees of intoxication are frequent and create a social and medical problem. The personal risks are: (1) He may die of exposure. (2) Alcohol in the tracheobronchial tree can cause pneumonia. (3) Inhale his vomit or dentures. (4) A bolus of food or meat inhaled into larynx can cause death due to choking. (5) He may fall and sustain a head injury. (6) He may fall into water and be drowned. (7) He may turn on the gas and forget to light the burners. (8) He may electrocute himself when handling with a plug or a defective electrical circuit. (9) He may take poison by mistaking it for alcohol. (10) An intoxicated person driving a motor vehicle is a danger to others. (11) The so-called **saturday night paralysis occurs in the stage of coma and results from pressure on a nerve trunk, as when an arm hangs over a chair (pressure on the radial nerve).**

Alcohol and Traffic Accidents: There is progressive loss of driving ability as blood alcohol concentration rises. 30 to 50 mg.% concentration of blood alcohol affects the driving ability of many persons.

In subdural haemorrhage due to head injury, if the person lives for some days, alcohol found in subdural haematoma at autopsy roughly corresponds to the alcohol level of the deceased at the time of head injury.

Alcohol after Death: Alcohol diffuses through the intact stomach wall after death into the surrounding blood and tissues, including the pericardial fluid and pleural fluid. As such, higher blood levels are found than what actually existed during life, if blood samples are taken from parts into which postmortem diffusion has taken place, i.e., heart or a large vein in the chest. Ethanol and other alcohols can be produced during the putrefactive process by fermentation of the carbohydrates and proteins of the body. Endogenous production of alcohol is seen in about 20% of cadavers. The longer the interval after death, and the higher the temperature, the more is produced. **Alcohol produced by advanced putrefaction may be as high as 200 mg.%. In such cases, alcohol will not be found in urine.**

METHYL ALCOHOL

Types: (1) **Pure methyl alcohol** (wood alcohol) is colourless, volatile liquid, with an odour similar to ethyl alcohol, and has a burning taste. (2) **Mineralised methylated spirit** consists of 90% by volume of ethyl alcohol, 9.5% of wood naphtha and 0.5% of crude pyridine. (3) **Industrial methylated spirit consists** of 95% by volume of alcohol and 5% of wood naphtha. It is present in certain home made beverages, antifreeze, paint removers, dyes, resins, adhesives and varnish.

Signs and Symptoms: Methyl alcohol produces symptoms of drunkenness in the same way as ethyl alcohol, but inebriation is not prominent, and the effects are more prolonged. Toxicity can result following its absorption through skin or respiratory tract. **Symptoms may appear within an hour or may not appear for 24 hours.** They consist of nausea, vomiting and pain or severe cramps in the abdomen, headache, dizziness, neck stiffness, confusion, vertigo.

There is marked muscular weakness and depressed cardiac action and hypothermia. There may be dyspnoea and cyanosis. **The odour is usually present in the breath. The effect on the central nervous system is more intense and persistent than with ethyl alcohol. There may be delirium and coma which may last for two or three days.** There is a toxic effect on the liver and kidneys (acute tubular necrosis) and on highly specialised nerve elements. Urine is strongly acid and may contain acetone and a trace of albumin. **An anion metabolic acidosis is characteristic of methanol, ethylene glycol and salicylate intoxication.** Anion gap acidosis is also seen in diabetic ketoacidosis and lactic acidosis. The pupils are dilated and fixed. **Visual disturbances like photophobia and blurred vision (snowfield vision),** seeing spots, central and peripheral scotomata, decreased light perception (flashes or snowstorm), concentric diminution of visual fields for colour and form, followed by **fairly sudden failure of vision or complete blindness occur due to optic neuritis and atrophy from the effect of formic acid on the optic nerve.** Fundoscopy shows hyperaemia of optic disc, followed by retinal oedema. The retinal ganglion cells, and optic disc show degenerative changes. In fatal cases convulsions are usual as a terminal event, and death occurs from respiratory failure. **An increased osmolol gap and anion metabolic acidosis accompanied by visual symptoms suggests methanol poisoning. Severe non-diabetic acidosis in unconscious persons is suggestive of methyl alcohol poisoning.**

Fatal Dose: 60 to 200 ml.

Fatal Period: 24 to 36 hours; may be delayed for 2 to 4 days.

Absorption: It is rapidly absorbed through the stomach and intestines, and also through the lungs and the skin. Though its action resembles that of ethyl alcohol to a great extent, **its rate of oxidation and elimination is one-fifth that of ethanol and with repeated small doses tends to accumulate in the blood. 80 mg/100 ml. of blood is dangerous level.** It does not completely disappear from the blood for three or four days. **Formaldehyde** (which is 33 times more toxic than methanol), **and formic acid are formed during the oxidation of methyl alcohol, which is responsible for the associated metabolic acidosis and the retinal toxicity.** It is distributed in the tissues according to their water content, and a high concentration is found in vitreous body and optic nerve.

Elimination: The liver slowly metabolises methanol. 3 to 5% is excreted through lungs and up to 12% through kidneys.

Cause of Death: Death is mainly due to acidosis from production of organic acids, and CNS depression is a minor factor.

Treatment: (1) Gastric lavage using 5% bicarbonate solution should be done and half litre of this may be left in the stomach. (2) **Activated charcoal** reduces the mortality significantly. It acts by reducing the absorption of alcohol from the digestive tract, and by creating a concentric gradient in favour of movement of alcohol and its metabolites back into the gut. (3) **Ethyl alcohol is the antidote.** It is given i.v. as a 10% solution, starting with 500 ml. as an infusion and repeated as required until blood level falls below 25 mg%. The i.v. route is preferred to avoid gastritis. Methyl alcohol is oxidised to formaldehyde by the enzyme catalase. This catalase can also oxidise ethyl alcohol to acetaldehyde. **In methyl alcohol poisoning, ethyl alcohol by competition for catalase, blocks the formation of formaldehyde and allows the less toxic methyl alcohol to be excreted unmetabolised.** (4) Alternatively 60 ml. of ethyl alcohol in 200 ml. fruit juice can be given orally over a period of 30 minutes. Give 15 ml. of 50% ethyl alcohol every hour for maintenance. (5) **4-methyl pyrazole (4-MP) or fomepazole.** The usual dose is 15 mg/kg followed 12 hours later by 10 mg/kg 12th hourly for 4 doses, and then increased to 15 mg/kg 12th hourly for as long as necessary. **It is a competitive inhibitor of alcohol dehydrogenase. It blocks formation of formaldehyde and formic acid and can be used instead of ethanol.** (6) **Folinic or folic acid** 50 to 75 mg. every four hours is useful to increase the elimination of formic acid, decreasing the metabolic acidosis and reducing symptoms. (7) **The basic treatment for alcoholic ketoacidosis is crystalloid therapy, dextrose, thiamine, and phosphate.** (8) Keep the airway clear. (9) Eyes should be kept covered to protect them from light. (10) **Haemodialysis** is the treatment of choice in severe poisoning. **It reduces the half-life of methanol**

from 40 hours to about one hour. There is no role for peritoneal dialysis or haemoperfusion. (11) Sodium bicarbonate i.v. to correct metabolic acidosis. (12) Blood sugar should be measured frequently while ethanol is being given, as it may cause hypoglycaemia, especially in children. (13) Symptomatic.

Postmortem Appearances: Cyanosis is marked and there is an absence of postmortem clotting of the blood. The pyridine may give the skin a purple colour. The mucous membrane of the stomach and the duodenum is congested and inflamed with small haemorrhages. **Small or large intestine or both are contracted resembling a thick pipe of very narrow lumen.** The lungs are congested and oedematous. The brain is oedematous and shows local haemorrhages. The mucosa of the bladder is often congested. The liver shows fatty change and sometimes early necrosis and there is tubular degeneration of the kidneys.

The Circumstances of Poisoning: Poisoning is mostly accidental. Sometimes, it is used as an intoxicating drink, when ethyl alcohol is not available.

ETHYLENE GLYCOL

It is a clear, colourless, odourless, non-volatile liquid with a bitter-sweet taste. It is mainly used as an antifreeze agent. It is not absorbed through skin. It is metabolised to glycoaldehyde, glycolic acid and oxalic acid and inhibits oxidative phosphorylation.

SYMPTOMS: Initial symptoms are vomiting, lethargy, ataxia, inebriation, convulsions, and coma. In 12 to 24 hours, tachycardia, tachyapnoea, and circulatory collapse occur. In 1 to 3 days, hypocalcaemia, oliguria, tubular necrosis, and renal failure occurs. Urine contains crystals of calcium oxalate.

FATAL DOSE: 100 to 200 ml.

FATAL PERIOD: 3 days

TREATMENT: (1) Gastric lavage. (2) Activated charcoal. (3) Ethanol in same dose as for methyl alcohol. (4) 4-methyl pyrazole. (5) Haemodialysis. (6) I.V. sodium bicarbonate. (7) 10% calcium gluconate i.v.

ISOPROPANOL

It is a colourless, volatile liquid with a faint odour of acetone and is slightly bitter. It is used as a disinfectant, paint remover, industrial solvent and for massage. It is absorbed through all routes. It is rapidly metabolised and converted to acetone which is excreted in urine and breath. It is two to three times more potent than ethanol as CNS depressant. Fatal dose is about 250 ml.

SYMPTOMS: Abdominal pain, gastritis, vertigo, headache, lethargy, ataxia, haemorrhagic tracheobronchitis, and apnoea.

TREATMENT: (1) Gastric lavage. (2) Activated charcoal. (3) Hemodialysis.

CHLOROFORM

It is a heavy, colourless, volatile liquid, with sweet pungent taste and a characteristic ethereal odour.

SIGNS AND SYMPTOMS: When swallowed there is burning pain in the mouth, throat and stomach and vomiting. Within minutes, unconsciousness and coma with slow stertorous breathing occurs. When inhaled, it causes irritation in throat and burning of eyes. The face is flushed and the patient becomes delirious. In 3 to 4 minutes, patient becomes unconscious and corneal and other reflexes are lost. Pulse and respiration are slow and feeble, temperature subnormal and pupils contracted. All the muscles are relaxed. If the inhalation is continued, the patient passes into a stage of paralysis. Death occurs from cardiac or respiratory failure.

FATAL DOSE: Thirty ml. when ingested; a concentration of 5% or more in air when inhaled.

TREATMENT: Stomach wash, artificial respiration, stimulants and symptomatic treatment.

POSTMORTEM APPEARANCES: They are not characteristic except the smell in serous cavities, lungs and brain. There is usually marked congestion.

THE CIRCUMSTANCES OF POISONING: Accidental death may occur during anaesthesia or when liquid chloroform is swallowed accidentally. It is extremely difficult to put a person under the influence of an anaesthetic without his consent.

ETHER

It is a colourless, volatile liquid having a peculiar penetrating odour and sweetish pungent taste.

SIGNS AND SYMPTOMS: When inhaled, effects are similar to chloroform, but there is more irritation of respiratory tract and more secretion of mucus and saliva. Taken by mouth effects are similar to alcohol, but is more rapid in onset and shorter in duration.

FATAL DOSE: 30 ml.

TREATMENT: As for chloroform.

POSTMORTEM APPEARANCES: Brain is slightly oedematous. Trachea contains frothy mucus. Lungs are congested and oedematous.

OPIUM

Characteristics: Opium (*afim*) is the dried juice of the poppy (*Papaver somniferum*) which is cultivated in India and other Eastern countries, only under a licence. The unripe capsule is incised and the white juice which exudes is collected and allowed to evaporate to obtain opium (Fig. 29-1). Ripe and dry poppy capsules contain a trace of

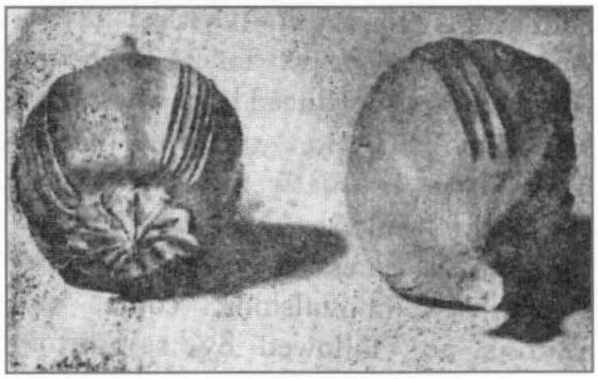


Fig. (29-1). Poppy capsules.

opium and are used for their sedative and narcotic action. Their warm decoction is used locally as sedative fomentation and poultice. **Poppy seeds** (*khas-khas*) **are white, harmless, demulcent and nutritive and are used as food** (Plate 23-142). The oil from the seeds is used for cooking purposes. **Opium** occurs in rounded, irregularly formed or flattened masses and has a strong characteristic odour and bitter taste. When fresh, it is soft and flexible and internally moist, coarsely granular or smooth and dark-brown on keeping.

Active principles: Crude opium contains a large number of alkaloids, about 25, combined with meconic, lactic, and sulphuric acids. These form two chemically different groups: (a) the phenanthrenes: morphine (about ten percent), codeine (about half percent), and thebaine (about 0.3%), which are narcotic, and (b) the isoquinoline group: papaverine (about 1%), and narcotine (about six percent), which have mild analgesic but no narcotic properties. Thebaine acts as a convulsant.

Morphine occurs as white powder or as white shining crystals, has bitter taste and alkaline reaction. The narcotic symptoms of opium poisoning are practically those of morphine poisoning.

Classification: (1) Natural: morphine, codeine. **(2) Semisynthetic:** Substance which are derived by modification of the chemical constituents of opium are called **semi synthetic narcotics**. They are chemically related to opium alkaloids and are called **opioid analogues**, e.g. heroin, hydromorphone, oxymorphone, oxycodone. **(3) Synthetic: Synthetic analogues called designer drugs are manufactured illicitly in clandestined laboratories for recreational use,** which are highly potent and addictive, e.g., meperidine, methadone, levarphanol tartrate, paregoric, diphenoxylate, fentanyl, propoxyphene.

Opioids are substances having action similar to opium but not derived from it.

Action: Opiates exert their effects because of their chemical similarity to natural substances called endorphins. The opiate drugs activate receptor sites normally occupied by natural opiates or endorphins. CNS is depressed with narcosis and respiratory inhibition. Opium depresses all centres, except oculomotor, vomiting and sweating.

Fatal Dose: Opium, 2 g; morphine, 0.2 g; codeine, half g; methadone, 100 mg; propoxyphene 1 g. and pentazocin 300 mg. Fentanyl is 50 to 100 times more potent than morphine.

Fatal Period: 6 to 12 hours.

Signs and Symptoms: The contact of morphine with the skin of sensitive persons may cause erythema, urticaria and itching dermatitis. When opium is taken by mouth, symptoms begin within half-hour. If the drug is injected, its action is noted within 3 or 4 minutes. It first stimulates, then depresses and finally paralyses the nerve centres.

(1) Stage of Excitement: There is a sense of well-being, increased mental activity, freedom from anxiety, talkativeness, restlessness or even hallucinations, flushing of face and greatly excited or maniacal condition may be seen.

(2) Stage of Stupor: The symptoms are headache, nausea, vomiting, incapacity for exertion, a sense of weight in the limbs, giddiness, drowsiness and stupor. The subject lies motionless, with eyes closed as if in a sound sleep from which he may be aroused at first, but soon passes into stupor and coma. The pupils are contracted, face and lips are cyanosed and an itching sensation is felt all over the skin. The pulse and respirations are normal.

(3) Stage of Coma: The patient passes into deep coma from which he cannot be roused. The muscles become relaxed and **all reflexes are abolished.** The face is pale, and conjunctivae congested. The pupils are contracted to pinpoint size and do not react to light, but in the late stage, and before death, they may be found dilated. **All the body secretions are suspended except sweat. Perspiration is very much increased.** Temperature is subnormal. Blood pressure is low, the pulse slow and full, the breathing is slow and stertorous and may be reduced to 3 to 4 per minute. The odour of opium may be present in breath.

In case of fatal termination, lividity of the surface increases, pulse becomes slow, irregular and imperceptible, the respirations become Cheyne-Stokes in type, and finally death occurs in deep coma from asphyxia.

DIFFERENTIAL DIAGNOSIS: (1) Opium poisoning: **The odour of breath, pinpoint immobile pupils, stertorous respiration, slow pulse and moist perspiring skin are prominent features. The triad of coma, immobile pupils and respiratory depression are diagnostic.**

(2) Acute alcoholic poisoning: **Odour of alcohol in breath, congested eyes and hyperaemia of face and neck, pupils dilated and reacting, subnormal temperature, slow and stertorous breathing.**

(3) Barbiturate poisoning: **Shallow respiration, deep coma, no response to painful stimuli, deep reflexes are depressed, subnormal temperature, low blood pressure, thready pulse, dilated pupils.**

(4) Carbolic acid poisoning: **Odour of breath, white patches on lips and mouth and carboluria.**

(5) Carbon monoxide poisoning: **History of exposure to poisoning with gas, intermittent convulsions, cherry-red colour of skin and carboxyhaemoglobin in blood.**

(6) Epileptic coma: **History of characteristic seizures, pupils fixed and dilated, skin flushed, face and lips cyanosed, froth at the mouth and nostrils, tongue may be bitten, slow respiration, rapid recovery.**

(7) Uraemic coma: **Gradual onset, pallor of face, Cheyne-Stokes respirations, ammoniacal odour, general anasarca, epileptiform convulsions, albumin, blood and casts in urine.**

(8) Diabetic coma: **Gradual onset, flushed face, deep respirations with air-hunger, low intraocular tension, odour of acetone, subnormal temperature, sugar and acetone in the urine.**

(9) Hysterical coma: **Usually in females, previous history with convulsive movements, usually occurs in presence of other persons, unusual attitude, reflexes are not altered.**

(10) Cerebral haemorrhage: **Old age, sudden onset, history of hypertension, slow full pulse, Cheyne-Stokes respirations, paralysis, usually hemiplegia, dilated pupils, raised temperature, bilateral extensor plantar reflexes.**

(11) Brain trauma: **History or evidence of head injury, bleeding from nose, mouth or ears, respirations rapid, irregular or Cheyne-Stokes, pulse rapid; later slow, pupils inactive, often unequal, paralysis of cranial nerves, subconjunctival haemorrhages.**

(12) Cerebral malaria: **History of fever with rigors, enlarged spleen, may be hyperpyrexia.**

(13) Encephalitis: **Acute onset, fever, involuntary movements, ocular palsies, changes in cerebrospinal fluid.**

(14) Meningitis: **Gradual onset, signs of meningeal irritation, fever, C.S.F. changes.**

(15) Heat hyperpyrexia: **Prolonged exposure to high temperature or sun, congested conjunctivae with contracted pupils, hyperpyrexia, absence of sweating and dry skin, circulatory collapse, and convulsions.**

Treatment: (1) Wash the stomach frequently with a solution of 1:5000 potassium permanganate, leaving some of the solution in stomach for oxidising the alkaloid. **Gastric lavage should be done even after hypodermic injection of the drug, for the alkaloid is re-excreted into the stomach after absorption.** (2) A tablespoonful suspension of activated charcoal may be introduced into the stomach. (3) Emetics usually fail due to the depression of vomiting centre. (4) The intestines should be cleared out by enema twice daily for two days to prevent reabsorption. 30 g. of sodium sulphate by mouth with large amounts of water is also helpful. (5) Establish adequate airway. Use endotracheal intubation if necessary. (6) **Atropine is not recommended**, for it can cause death by paralysing the motor and sensory nerves just like morphine. (7) **Nalorphine and levallorphan are not recommended.** (8) **Naloxone hydrochloride is a specific opioid antagonist.** Unlike levallorphan and nalorphine, it neither causes psychomimetic effects nor CNS depression. **It competes with opioids at receptor sites.** It reverses the depressant, analgesic and euphoric effects of opioids and also the dysphoric, delusional and hallucinatory properties of the synthetic opioids. 2 mg. is given i.v. if there is respiratory depression, and repeated every half to one hour up to a total dose of 10 to 20 mg. It can also be given i.m. or sublingually. Both coma and cardiopulmonary depression are reversed. If there is no response in two minutes, endotracheal intubation is necessary, and the same dose should be repeated. In any opioid overdose, a continuous infusion at a rate of 0.4 to 0.8 mg per hour may be continued up to 48 hours, especially in long-acting opioids, such as methadone. (9) **Nalmefene has longer duration of effect than naloxone.** 0.1 mg is given i.v. followed by one mg in 2 to 5 minutes. It can be given i.m. or s.c. (10) When coma is deep, artificial respiration and oxygen is given by inhalation. (11) Physostigmine 0.04 mg/kg i.v. may

be given to reverse respiratory depression if naloxone is not available. (12) Symptomatic. (13) **Coma cocktail:** In comatose patients where the identity of poison is not known, 100 ml. 50% glucose, 100 mg thiamine and 2 mg naloxone should be given i.v. (14) If the patient is seen in the early stage, he should be made to walk about in the open air to help excretion, but if poison has been absorbed and is acting upon the cells of the cortex, it may do more harm than good by further exhausting the patient. (15) Buprenorphine and LAAM are considered effective in long-term management. (16) Symptomatic.

Postmortem Appearances: They are not characteristic, but **signs of asphyxia are prominent.** The face and the nails are cyanosed. Froth is seen at the mouth and nostrils. Postmortem staining is well marked and cyanotic. The smell of opium is noticed on opening the chest, but it disappears if putrefaction has set in. The stomach may contain small lumps of opium. The trachea and bronchi are congested and covered with froth. The lungs are oedematous and congested. The blood is usually dark and fluid. The brain, meninges and abdominal organs are congested. **Opium disappears rapidly from the cadaver.**

Absorption: It is absorbed from mucous membranes, raw surface or wounds, hypodermic injection and when smoked in cigarettes.

Elimination: It is destroyed by the tissues and particularly by the liver. It is eliminated mainly as morphine in urine and faeces, and also by stomach, intestines, saliva, bile and milk.

Poisoning: **(1)** Opium is selected by suicides because death is painless. (2) Homicide is rare. (3) Poisoning may occur in addicts. (4) Drugging of children by opium to keep them quiet, may result in accidental poisoning. (5) Sometimes, opium is used to steady the nerves for doing some bold act. (6) It is rarely used as cattle poison. It is sometimes used for doping race horses.

CHRONIC POISONING (MORPHINISM; MORPHINOMANIA): The mechanism of tolerance is not known, but is thought to reside at a cellular level. The habit is acquired by young people as it is believed to be an aphrodisiac, and as it produces a sense of euphoria. Opium addicts can tolerate 3 to 6 g. per day. The morphine addict has a dry skin, and shows scars of healed abscesses or abscesses themselves, and sometimes tattooing from needles. Unusual mood swings, periods of depression alternating with euphoria occur. The patient becomes restless and irritable and sleep is disturbed by dreams or there is insomnia. Loss of memory, mental fatigue and gradual intellectual and moral deterioration occur. Constipation, contracted pupils, anorexia, emaciation, sweating, tremors, and weakness and impotence are frequent.

MEPERIDINE (PETHIDINE): Meperidine hydrochloride is a colourless, crystalline powder with a bitter taste. It is administered by the i.m. or i.v. route for its analgesic, antispasmodic and sedative properties.

Action: It acts on the cerebrum and produces analgesia and sedation.

Fatal dose: About 2 g.

Fatal period: 24 hours.

Symptoms: Effects are similar to those of morphine. It causes more dizziness than morphine and greater elation. Tolerance to its toxic effects is not complete, and addicts may have twitchings, tremors, mental confusion, hallucinations, dilation of the pupils, dry mouth and sometimes convulsions. The impairment of ability to work is more than with morphine. Abstinence syndrome resembles that of morphine withdrawal and symptoms appear in 3 to 4 hours and reach maximum intensity 8 to 12 hours later. Then they decline rapidly and disappear in four to five days.

It is a drug of addiction. The addiction is common among doctors and nurses. Monoamine oxidase inhibitors and phenothiazines can produce severe reactions and even death when taken together with pethidine.

Treatment: Same as for opium.

Opioids: Other opioid drugs of addiction include: codeine, dihydrocodeine, papaverine, pethidine, methadone, dipipanone, dextromoramide, pentazocine, cyclizine, diphenoxylate and dextropropoxyphene.

HEROIN (BROWN SUGAR): There are three types of heroin, white, brown and black tar. Street heroin is known as "smack, junk, or dope" and is diluted with quinine, lactose, mannitol, etc. **It is the most dangerous among all drugs of addiction.** It can be smoked or injected, or used as snuff. **A combination of heroin and cocaine is known as "Speedballs". Fatal dose is about 50 mg.**

It is metabolised to monoacetylmorphine or acetylmorphine. Monoacetylmorphine is then hydrolysed to morphine (half-life 38 minutes). As such, chemical analysis will detect morphine but not heroin. After injection, morphine and monoacetylmorphine are found in the urine almost immediately. **Tolerance occurs very rapidly (within days) and can be increased to more than hundred times the initial dose.** Intense euphoria lasts for several minutes followed by sedation for about one hour, and the effects are completely lost in 3 to 6 hours. It causes excitation by lifting cortical inhibitions similar to alcohol. **It can cause sudden death, even in persons who have been using it for some time, with the needle still in the vein.** A few deaths may be due to some personal hypersensitivity to the drug. **Fatal dose is 50 mg.** Overdose causes death from a very strong CNS depressant action. In almost all cases, the victims are under the influence of alcohol at the time of death.

Treatment: (1) **Methadone 40 mg daily** will usually prevent withdrawal symptoms. In a chronic addict, 80 mg. will usually prevent heroin-induced euphoria. The dose is gradually reduced by 20% daily. If signs of withdrawal appear, dose reduction should proceed more slowly. Heroin addicts should never be given 20 mg. methadone at one time. (2) Detoxification. (3) Narcotic antagonist, such as naltrexone, naloxone, haloperidol, clonidine and cyclazocine.

Autopsy: Lungs are heavy and congested. Severe pulmonary oedema is the common autopsy finding probably due to sudden ventricular dysrhythmia. Microscopically, lungs show foreign body granulomas. Liver shows chronic triaditis with mononuclear cell infiltrates.

SEDATIVE-HYPNOTICS: Sedative drugs are those that decrease activity, moderate excitement and exert calming effect. A hypnotic drug produces drowsiness and facilitates a state of sleep, resembling natural sleep.

BARBITURATES

They are white crystalline, odourless powders, with a faintly bitter taste.

Action: They have a depressant action on CNS. Large doses directly depress the medullary respiratory centre.

CLASSIFICATION: (1) Long-acting: (onset of action two hours and duration of action 6 to 12 hours) barbitone, phenobarbitone, methyl phenobarbitone, diallylbarbituric acid, mephobarbital, phenytoin.

(2) Intermediate-acting: (onset of action half to one hour and duration of action 3 to 6 hours): amylobarbitone, butobarbitone, probarbitone sodium, aprobarbital, vinbarbital, allobarbitone.

(3) Short-acting: (duration of action 3 hours): cyclobarbital, pentobarbital, seconal, ortal, amobarbital, cyclobarbitone, quinalbarbitone.

(4) Ultra-short acting: (onset of action immediate and duration of action 5 to 10 minutes): pentothal sodium, hexobarbital sodium, kemithal sodium, and thiamylal sodium.

ABSORPTION, DISTRIBUTION AND ELIMINATION: They are rapidly absorbed from the GIT including the rectum, and from the subcutaneous tissues. They are concentrated in the liver for a short time and then evenly distributed in the body fluids. They are partly destroyed in liver and excreted in urine. Barbiturates, alcohol and CO produce irreversible brain damage and yet the patient survives for a sufficiently long period so that they are completely metabolised or excreted before death occurs.

Signs and Symptoms: Acute poisoning may result from a single large dose, or with repeated small doses. **Usually the first symptom is drowsiness.** A short period of confusion, excitement, delirium, and hallucinations is common. Ataxia, vertigo, slurred speech, headache, paraesthesias and subjective visual disturbances occur. A stupor progressing to deep coma, with inhibition or loss of superficial and deep reflexes and gradual loss of response to painful stimuli occur. The Babinski toe sign may become positive. Respirations may be rapid and shallow or slow and laboured, but the minute volume is always reduced. There is a fall in cardiac output and an increase in capillary permeability leading to an increase in the extracellular fluid. **Mild but progressive cardiovascular collapse occurs.** The pupils are usually slightly contracted but react to light; they may dilate during terminal asphyxia. The urine may be scanty or suppressed and may contain sugar, albumen and haematoporphyrin. Incontinence of urine and faeces may occur. The body temperature is usually reduced; fever indicates bronchopneumonia. Respirations become irregular, sometimes Cheyne-Stokes in character and finally stop. **Blisters on the skin, often on an area of erythema, strongly**

suggest barbiturate poisoning. Blisters contain clear serous fluid. Rupture of a blister can be mistaken for a burn. Blisters are commonly found in sites where pressure has been exerted between two skin surfaces, such as the interdigital clefts and inner aspects of the knees. Occasionally, an entire side of a forearm or a thigh is blistered. The coma may continue for a few hours to a few days, and the patient then makes a gradual recovery. Death may occur from respiratory failure or ventricular fibrillation in early stages and bronchopneumonia or irreversible anoxia with pulmonary oedema in the late stages. **The combination of alcohol and barbiturates causes rapid death.** Phenytoin causes gingival hyperplasia in chronic therapy.

Fatal Dose:

Short-acting: 1 to 2 g.

Medium-acting: 2 to 3 g.

Long-acting: 3 to 5 g.

Fatal Period: 1 to 2 days.

Treatment: (1) Gastric lavage should be carried out up to 8 hours post-ingestion, with warm water mixed with potassium permanganate and suspension of activated charcoal or tannic acid. A concentrated solution of magnesium sulphate should be left in the stomach. (2) **There is no specific antidote.** Analeptic therapy should be avoided unless a clear and compelling need exists. (3) **Scandinavian method, uses antishock measures, maintenance of patent airway, and adequate respiratory support. CNS stimulants have been totally eliminated.** Fluid replacement therapy should be used and not vasopressors. If shock persists dopamine should be given. (4) Normal saline with 5% glucose i.v. increases the rate of excretion. (5) Artificial respiration and oxygen. (6) The patient should be kept warm and mucus removed from the throat. (7) Bowels should be evacuated by enema. (8) Noradrenaline two mg. diluted with half litre of 5% glucose in saline i.v. to counteract shock and low blood pressure. (9) Haemodialysis and exchange transfusion are sometimes life-saving. (10) Forced alkaline diuresis is most useful in poisoning by barbiturates which are not protein-bound like phenobarbitone, allobarbitone and barbitone. (11) Charcoal haemoperfusion. (12) Antibiotics to minimise risk of pneumonia. (13) Symptomatic.

Postmortem Appearances: They are not characteristic, but are mainly those of asphyxia. Cyanosis is present. A quantity of white particles of ingested barbiturate may be seen in the stomach with mucosal congestion or mild corrosion. The lungs are congested, oedematous and pneumonic. Petechial haemorrhages may be present in the lungs and on the pleura and pericardium. The kidneys show degeneration of the convoluted tubules. Other organs are congested.

CHRONIC POISONING: Tolerance develops rapidly and is usually marked. There is also a cross-tolerance with alcohol. It occurs when barbiturates are used therapeutically in epilepsy or psychoneurotic patients. The signs and symptoms resemble those of chronic alcoholism with progressive impairment of cerebral function, dysarthria, ataxia, and depression. Tendon reflexes may be depressed or there may be hypertonia and tremors of Parkinsonian type. Addiction is a serious problem. Dependence is both psychic and physical. The impairment of mood, behaviour, and intellectual functions causes social deterioration. Withdrawal symptoms appear within a day and may persist for two weeks. Common features are: anxiety, nausea, vomiting, weakness, hypotension, tremors, and disturbances of vision. Convulsions may occur.

Poisoning: (1) They are used for committing suicide. (2) Rarely used for homicide. (3) Accidental poisoning may result due to "automatism".

METHAQUOLONE: "Mandrax" and "melsedine" containing methaquolone are used for insomnia. Some persons are extremely sensitive to this drug and may become unconscious even after one tablet. If it is taken some hours after food, the patient may feel dizzy and sweats and a syndrome similar to hypoglycaemia is produced. 2.5 g. of the drug produces unconsciousness within half hour. Addiction may occur and some degree of tolerance is seen after prolonged use.

CHLORAL HYDRATE (Dry Wine)

It is colourless, crystalline substance having peculiar pungent odour and a pungent bitter taste. It depresses CNS. It is absorbed rapidly from the stomach and small intestine, and also from the rectum.

Signs and Symptoms: They resemble those of barbiturates, but in addition there is retrosternal burning sensation, vomiting, and rarely jaundice. Sometimes due to idiosyncrasy, a scarlatinal or urticarial rash may be seen on the skin. Death usually occurs from paralysis of the respiratory centre.

Chronic Poisoning: Symptoms are those of gastrointestinal irritation with erythematous and urticarial eruptions on skin, tremors, and dyspnoea. Convulsions, mental degeneration, and liver damage may occur. Habitual use can lead to tolerance and physical dependence with delirium when the drug is withdrawn.

Fatal Dose: 5 to 10 g.

Fatal Period: 8 to 12 hours.

Treatment: (1) Wash the stomach with alkaline solution. (2) Forced diuresis or dialysis. (3) Flumazenil 0.1 mg as infusion to a total of 3 mg. (4) Symptomatic.

Postmorten Appearances: Gastric mucosa is softened, reddened and eroded and smells of chloral hydrate. Brain and lungs are congested. Hepatorenal damage is seen.

Poisoning: (1) Accidental poisoning results by taking large doses as hypnotic. (2) Suicidal cases are rare. (3) **It is given in food or drink to render a person suddenly helpless for the purpose of robbery or rape. Its action is so rapid that it has been given the name of "knockout drops", A combination of alcohol and chloral is commonly known as "Mickey Finn".** (4) It is often added to liquor to increase its potency.

BROMIDES

SYMPTOMS: There may be nausea, colic and vomiting due to the local action, followed by vertigo, staggering gait, mental confusion, slurred speech, tremors, constricted pupils, fall of blood pressure, feeble pulse, subnormal temperature, shallow respirations, muscular weakness or paralysis, collapse, stupor or coma. The bromides displace chlorides from the plasma and cells, and may cause fatal depression of the nervous system.

FATAL DOSE: 30 to 45 g.

FATAL PERIOD: 6 to 18 hours or more.

TREATMENT: (1) Gastric lavage. (2) Chloride hastens the elimination of bromides, and as such normal saline i.v. by drip method or 2 g. of sodium chloride in capsules every four hours by mouth are useful. (3) Symptomatic.

HYDROCARBONS

Most of the hydrocarbons are derivatives of petroleum distillates. The toxic substances like gasoline, kerosene, naphtha, mineral spirit, light gas oil, and mineral seal oil are poorly absorbed from the GI tract. **Benzene, toluene and xylene are highly volatile and well absorbed from the GI tract. Methane and butane are gases and act as simple asphyxiants.** Lubricating oil and asphalt are non-toxic. Turpentine and pine oil are readily absorbed from the GIT.

Signs and Symptoms: Acute or chronic contact with hydrocarbons causes chronic eczematoid dermatitis, with redness, itching and inflammation. Cutaneous exposure to gasoline and other hydrocarbons can cause second degree burns, and systemic manifestations. When ingested, symptoms are: nausea, vomiting, abdominal pain and diarrhoea with risk of aspiration pneumonitis. The breath, vomit and urine have the peculiar smell. The pupils are first contracted, but become dilated when coma supervenes. Aspiration produces coughing, choking, gasping, bronchospasm, hypoxia and CNS depression. Haemoptysis may be present in severe cases. Haemorrhagic pulmonary oedema with pink, frothy sputum may soon progress to shock and cardio-respiratory arrest. Toluene sniffing causes drunken appearance.

Fatal Dose: 30 to 100 ml. of kerosene.

Fatal Period: Within one day.

Treatment: (1) Remove contaminated clothing, and wash the affected areas of skin with soap and water. (2) Syrup of ipecac to produce vomiting. (3) Gastric lavage with warm water containing sodium bicarbonate. (4) If inhaled, the patient must be taken into the open air, artificial respiration started and oxygen given. (5) Keep the airway patent. (6) Absorption of ingested kerosene can be slowed by giving 250 ml. of liquid paraffin orally.

Postmortem Appearances: Signs of asphyxia are present. There may be acute gastroenteritis and the odour may be present in the lungs and alimentary canal. The lungs are heavy, firm and airless. Petechial haemorrhages into the mucous membrane and subserous tissues may be found in the trachea, gastrointestinal tract and elsewhere. **In toluene poisoning, red cells may show basophilic stippling.**

Poisoning: Poisoning is usually accidental, especially among children. In the siphoning of gasoline from a tank, the mobile liquid can easily be aspirated in the lungs and cause death.

PARALDEHYDE

This is a clear, colourless liquid with an unpleasant ethereal odour, and an acrid nauseous taste.

SIGNS AND SYMPTOMS: When ingested, there is nausea, vomiting, eructations, headache and giddiness, and the breath smells of paraldehyde. Drowsiness sets in, pulse becomes slow and feeble, shallow breathing, dilated pupils, cyanosis, subnormal temperature, coma and death may occur from respiratory failure. Pulmonary oedema and bronchopneumonia are common.

FATAL DOSE: 25 to 50 ml.

FATAL PERIOD: Few hours.

TREATMENT: (1) Gastric lavage with a solution of sodium bicarbonate to be continued until washings cease to smell of the drug. Activated charcoal is useful. (2) High colonic irrigation. (3) Calcium gluconate and dextrose intravenously. (4) Artificial respiration and oxygen inhalation. (5) Symptomatic.

POSTMORTEM APPEARANCES: The body smells of paraldehyde. The mucosa of the stomach is hyperaemic and may be slightly inflamed. The viscera are congested. Pulmonary oedema and bronchopneumonia are usually seen.

CHRONIC POISONING: Symptoms are similar to those seen in chronic alcoholism, such as gastric irritation, muscular weakness, and tremors of the hands and tongue. Hallucinations and delusions may be present.

TURPENTINE

It is obtained by the distillation of the oleoresin from various species of pine tree.

When fresh it is colourless. It is extensively used to dissolve varnish paint and grease stains. It is eliminated from the lungs, and breath has a characteristic smell. In the urine it is excreted in combination with glycuronic acid. Some amount is excreted by the skin.

FATAL DOSE: 150 to 200 ml.

FATAL PERIOD: Few minutes to 12 hours.

SIGNS AND SYMPTOMS: SKIN: Redness, itching vesication. G.I.T: Nausea, vomiting, colic diarrhoea. R.S.: Central depression, pneumonia. C.N.S.: Excitement, giddiness, delirium, convulsions, coma, Renal: albuminuria, haematuria, oliguria, renal failure.

TREATMENT: (1) Gastric lavage with weak solution of sodium bicarbonate, (2) demulcents, (3) supportive therapy.

POSTMORTEM APPEARANCES: Stomach shows haemorrhagic spots and contents smell of turpentine. Lungs, brain and meninges are congested. Kidneys show degenerative changes.

POISONING: (1) Accidental. (2) Suicidal. (3) Abortificient.

CHAPTER

30 Psychotropic Drugs

Psychotropic drugs are substances that affect psychic function, behaviour or experience. They consist of: (1) Antidepressants. (2) Neuroleptics. (3) Hallucinogens.

The toxicity of the antidepressants is due to (1) their anticholinergic effects such as: supraventricular tachycardia, agitation, seizures, coma, hallucinations, and respiratory depression, (2) their ability to block reuptake of norepinephrine at the synapses, resulting in both atrial and ventricular disturbances and hypertension, (3) their quinidine-like membrane depressant effects on the heart by altering sodium influx resulting in conduction delays and myocardial depression, (4) peripheral alpha blockade causing hypotension, and (5) inhibition of sympathetic reflexes centrally.

CYCLIC ANTIDEPRESSANTS

SYMPTOMS: **Dry mouth, blurred vision, myoclonic jerks, dilated pupils, drowsiness and agitation are common symptoms. There may be cardiac arrhythmias, hypotension, convulsions and respiratory depression.**

TREATMENT: **(1) Stomach wash (up to 24 hours). (2) Physostigmine. (3) Sodium bicarbonate i.v. (4) Symptomatic.**

FATAL DOSE: **2 to 5 g.**

STIMULANTS: **They include amphetamine, cocaine, methamphetamine, caffeine and methylphenedate. These agents stimulate CNS, induce sense of wellbeing, increase alertness, decrease fatigue, suppress appetite and can lead to strong psychological dependence.**

MONOAMINE OXIDASE INHIBITORS

ACTION: **They block action of monoamine oxidase, resulting in alterations of neurotransmitter metabolism.**

SYMPTOMS: **They appear after 12 hours. Headache, tremors, confusion, convulsions, hallucinations, hyperpyrexia, hypertension followed by hypotension, coma, cardiovascular collapse.**

TREATMENT: **(1) Stomach wash. (2) Symptomatic.**

FATAL DOSE: **2 to 5 mg/kg.**

METHAMPHETAMINE: **Action:** Amphetamines increase the synaptic concentration of neurotransmitters dopamine and norepinephrine. They are powerful stimulants of CNS and CVS. It may be taken orally, i.v., smoked or sniffed. The onset of fatigue is delayed and tasks are more easily completed, but there is a loss of judgment and accuracy.

Symptoms: Acute poisoning: Symptoms are similar to cocaine poisoning.

(1) Mild: Restlessness, talkativeness, insomnia, tremors, sweating, dilated pupils.

(2) Moderate: Hyperactivity, confusion, hypertension, tachycardia, tachypnoea, vomiting, sweating, hallucinations.

(3) Severe: Delirium, hyperpyrexia, convulsions, coma, arrhythmias.

Acute intoxication may present as a paranoid hallucinatory syndrome which closely mimicks paranoid schizophrenia.

Designer drugs of amphetamine are abused by youngsters during rave parties for dancing throughout the night.

Chronic poisoning: (1) Amphetamine psychosis is characterised by: (a) Stereotyped, compulsive behaviour, (b) paranoid personality, (c) delusions, usually, persecution, (d) Hallucinations, usually visual, sometimes tactile. (2) Cardiomyopathy. (3) Intracranial haemorrhage.

Treatment: (1) Gastric lavage. (2) Acidification of urine. (3) Symptomatic. (4) Chlorpromazine for amphetamine psychosis.

Fatal Dose: 1 to 2 g.

P.M. appearances are those of asphyxia.

M.L. imp: Long-term use leads to psychological dependence and tolerance.

Dangers of misuse are: (1) Overactivity or aggressive behaviour. (2) Paranoid psychosis. (3) Shock and collapse. (4) Risk of suicide during the withdrawal phase.

ANTIPSYCHOTIC AGENTS (TRANQUILISERS)

ACTION: **The phenothiazines are anticholinergics and antidopaminergics to varying degrees. Other**

properties are central and peripheral cholinergic blockade, and adrenergic action secondary to the inhibition of reuptake of catecholamines.

SYMPTOMS: Myocardial depression, hypothermia or hyperthermia, decreased sweating and salivation, amenorrhoea, miosis, decreased intestinal motility and secretions, agranulocytosis, haemolytic anaemia, ventricular tachycardia, orthostatic hypotension, sedation, seizures, gynaecomastia, corneal opacities, cholestatic jaundice, priapism, laryngospasm, urticaria, dermatitis, photosensitivity and gray-blue pigmentation. There are three acute movement disorders occurring between 1 day and 60 days of initiation of therapy. (1) Acute dystonia: oculogyric crisis, jaw, tongue, lip and throat spasms, neck twisting, opisthotonos, facial grimacing and abdominal wall spasm. Symptoms rapidly resolve with parenteral antihistamines, anticholinergics, or benzodiazipines. (2) Akathisia: restlessness and inability to sit. (3) Parkinsonism: shuffling gait, resting tremors, rigidity, pill rolling, a mask-like expression, fine movements, muscle weakness, and bradykinesia are typical symptoms.

TREATMENT: (1) Emesis. (2) Gastric lavage. (3) Activated charcoal in repeated doses. (4) Catharsis. (5) Symptomatic.

BENZODIAZEPINES

The commonly used preparations are: Diazepam, flurazepam, chlordiazepoxide, nitrazepam, oxazepam, flurazepam, alprazolam, and lorazepam. They are tranquilisers commonly used to relieve anxiety. Absorption from GI tract is slow. Excretion in the urine may continue for several days. Addiction may occur.

Fatal Dose: 100 to 300 mg/kg body weight. **If taken alone they are not toxic, but mixed with alcohol or other drugs, they can contribute to death.**

Signs and Symptoms: Acute poisoning causes diplopia, dysarthria, ataxia, sedation and somnolence.

Chronic Poisoning: High dose, long-term therapy (30 to 40 mg of diazepam daily) may produce withdrawal symptoms when stopped suddenly, such as: **C.N.S.:** headache, anxiety, insomnia, muscle spasms, tremors, rarely convulsions and psychiatric disturbances. **G.I.:** anorexia, vomiting. **R.S.:** respiratory depression is rare.

Treatment: (1) Gastric lavage. (2) Activated charcoal. (3) Flumazenil selectively blocks the central effects by competitive interaction at the benzodiazipine recognition site. It is given in a dose of 0.2 mg/min as infusion to a total of 3.5 mg. If resedation occurs (in 20 to 120 minutes), the dose is repeated. (4) Forced diuresis. (5) Intensive supportive therapy.

PSYCHEDELICS (HALLUCINOGENS)

Psychedelics are substances that produce an alteration in environmental awareness while the individual maintains the capacity to recognise that what he is experiencing is not real. Such person is usually fully awake, alert and oriented but confronted with varied perceptual abnormalities and varied sensations. Sensory perception and thought processes are grossly distorted, and mood is altered. Reality (space, time, bodily dimensions) is distorted and a feeling of depersonalisation makes the person feel separated from the situation. Hallucinations produced by drugs, usually have some environmental stimulus providing the basis for the illusion. Most of the hallucinations are visual. Often hallucinations take the form of gross somatic distortions. Occasionally, blending of sensory modalities (synesthesia) may be noted. Profound philosophical thoughts, deep introspection, self-examination or religious overtones may be prominent components of the psychedelic experience. The response to a psychedelic is related to the persons mindset, emotions, or expectations at the time and can be altered by the setting. The person may experience euphoria or dysphoria, can be emotionally labile but usually realises that he is under the influence of a drug.

LSD, cannabis, mescaline, dimethyl tryptamine (DMT), psylocybin, psylocin, peyote, and phencyclidine (PCP) are important hallucinogenic drugs. **Hallucinogenic mushrooms** or "magic mushrooms" contain psilocybin and psilocin and are taken orally, raw or cooked.

Action: They involve various neurotransmitters in the CNS. LSD involves the serotonin system and tropane alkaloids (atropine, scopolamine, and hyoscyamine), have anticholinergic effects. There is psychic dependence only and no abstinence syndrome.

Signs and Symptoms: Both sympathetic and parasympathetic symptoms are produced. Sympathetic symptoms may include dilated pupils, tachycardia, tachyapnoea, hyperthermia, diaphoresis, piloerection, dizziness, weakness, hyperactivity, muscle weakness, ataxia, altered mental status and coma. Parasympathetic symptoms include salivation, lachrymation, diarrhoea, nausea, vomiting, bronchoconstriction and hypertension.

During hallucinations, **sensory perceptions are intensified: colours seem brighter and more clear, sounds seem excessively loud with an exaggeration of detail. The individual feels a sense of depersonalisation and separation from the environment. The person may perceive that he is observing an event as opposed to being involved in one.** The person's body image may become distorted, so also the boundaries of objects in the environment. **Alternatively, synesthesias or sensory misperceptions occur such as hearing colours or seeing sounds.**

Drugs that may alter mood, such as benzodiazepines, barbiturates, and amphetamines can produce perceptual changes during withdrawal. Alcohol in excess (alcoholic hallucinosis), or alcohol deprivation (delirium tremens) can cause hallucinations.

Lysergic acid diethylamide (LSD): It is a colourless, tasteless, odourless, semi-synthetic compound, the lysergic acid portion of which is a natural product of the ergot fungus Claviceps purpurea. **The dose required to produce psychotropic effects (take a trip) is 100 to 200 µg.** The trip usually occurs after half to one hour, peaking after 2 to 6 hours and fading after 12 hours. The effects depend very much on the individual and on the circumstances, with the same user having a bad or good "trip" on different occasions or even within the same trip. Symptoms are dry month, sweating, dilated pupils, tachycardia, hypertension, tremors, extreme mood changes, visual hallucinations, alteration in time perception, etc. Altered changes in vision and hearing like floating feeling, illusions, sensation of synesthesia or seeing smells or hearing colours occur. The action may persist for 12 to 18 hours. Tolerance develops rapidly. It is commonly taken as: (1) liquid on sugar, (2) saturated sugar cube, (3) soaked into blotting paper, (4) capsule, and (5) blue pills. Repeated use by an addict can lead to permanent psychosis.

Fatal Dose: About 14 mg.

The after-effects may persist for days or weeks. At the height of the effects of the drug on the mind, individual becomes violent or panic-stricken and he may attack others (urge to kill) or hurt himself from disregard of reality, and the normal considerations of the safety. In some persons lasting disturbances, depersonalisation, chronic dread, depression, mood swings and paranoid attitudes and belief may be found following repeated exposure to LSD. **The feeling of being able to fly under the influence of LSD can lead users to jump out of windows.** Biological halflife of LSD in man is three hours. Tolerance develops fast and also disappears quickly. Repeated use by an addict can lead to permanent psychosis.

Flashback Phenomenon: This may occur days, weeks or even months after the ingestion of a dose, and **the person experiences a recurrence of the emotional and psychological aspects of the previous "LSD" trip. These delayed recurring symptoms may lead to eccentric behaviour, suicide or even homicide.**

Treatment: (1) Antipsychotics. (2) Benzodiazipines. (3) Psychotherapy.

CHAPTER

31 Deliriant Poisons

DATURA FASTUOSA

Characteristics: There are two varieties: (1) Datura alba, a white-flowered plant, and (2) Datura niger, a deep-purple flowered plant. It grows on waste places all over India. The fruits are spherical and have sharp spines (thorn-apple) and contain yellowish-brown seeds (Table 31-1 and Plate 23-137 and 24-148). The flowers are bell-shaped. Datura stramonium grows at high altitudes in Himalayas.

Active Principles: **All parts of these plants including nectar (honey) are poisonous, especially the seeds and the fruit. They contain 0.2 to 1.4% of hyoscine (scopolamine), hyoscyamine and traces of atropine.**

Action: The alkaloids first stimulate the higher centres of brain, then the motor centres and finally cause depression and paralysis, especially of the vital centres in the medulla.

Signs and Symptoms: Contact with leaves or flowers cause dermatitis in sensitive persons. If the seeds are eaten, symptoms appear within half hour, if a decoction of the seeds is given within a few minutes, and if alkaloids are used almost immediately. A bitter taste, dryness of mouth and throat, with difficulty in talking, dysphagia, burning pain in the stomach and vomiting are first noticed. The voice becomes hoarse. The face becomes flushed, conjunctivae congested, pupils widely dilated with loss of accommodation for near vision, developing in temporary blindness, photophobia and diplopia. Light reflex at first is sluggish and later absent. The pollen can cause unilateral mydriasis (corn-picker's pupil). Mental changes include restlessness and agitation, and patient cannot recognise relatives or friends. Urinary retention and inability to pass urine occurs. The patient becomes confused, giddy, staggers as if drunk. The skin is dry and hot, the pulse rapid, 120 to 140 per minute, full and bounding, but later becomes weak and irregular, and the respirations are increased. The temperature may be raised by 2 or 3 degrees. Muscle tone and deep reflexes are increased and there may be muscular spasm. A scarlatinal rash or exfoliation of the skin may be seen over most of the body. Delirium is **restless and purposeless; in its earlier stages it is indicated by excitement, talkativeness and unintelligent speech. The patient may be silent, but**

Table (31-1). Difference between the seeds of datura and capsicum

Trait	Datura seeds	Capsicum seeds
(1) Size :	Large and thick.	Small and thin.
(2) Shape :	Kidney-shaped.	Rounded.
(3) Surface :	Numerous small depressions.	Smooth.
(4) Colour :	Dark or yellowish-brown.	Pale-yellow.
(5) Margins :	Laterally compressed and double-edged at the convex border.	The convex border is simple and sharp.
(6) Smell :	Odourless.	Pungent.
(7) Taste :	Bitter.	Pungent.
(8) Embryo :	On longitudinal section, embryo is curved outwards at the hilum.	Embryo is curved inwards like figure 6.

usually he is noisy, tries to run away from his bed, picks at the bed clothes, exhibits typical pill rolling movements, tries to pull imaginary threads from the tips of his fingers, threads imaginary needles. Hallucinations of sight and hearing and delusions occur. As intoxication advances, this excitement passes off in one to two hours and the patient passes into deep sleep or coma which may end rarely in death from respiratory paralysis. The patient may remain in this condition for two to three days, but usually distinct improvement occurs in 24 hours.

8 Ds: Dryness of mouth, dysphagia, dilated pupils, dry hot skin, drunken gait, delirium, drowsiness, death due to respiratory failure.

Fatal Dose: 1 g. (100 to 125 seeds). Atropine 120 mg; hyoscine 30 mg.

Fatal Period: One day.

Treatment: (1) Emetics can be used. (2) Wash out the stomach repeatedly with a weak solution of tannic acid. (3) Give activated charcoal and a cathartic. (4) Wash out the lower bowel frequently. (5) **Physostigmine** one mg i.v. or i.m. In many cases a single dose is sufficient. (6) **Pilocarpine** nitrate, 5 mg. s.c. is useful. It can be repeated after two hours. (7) Delirium can be controlled by bromides and short-acting barbiturates. (8) Symptomatic.

Postmortem Appearances: They are not characteristic, but are those of asphyxia. Seeds or their fragments may be found in the stomach and intestines. The stomach may show slight inflammation and the lungs oedema. The seeds resist putrefaction for a long time.

The Circumstances of Poisoning: (1) **Crushed or powdered seeds or an extract is used by criminals for stupefying a victim prior to robbery, rape** or **kidnapping (road poison).** It is usually given in food or drink, e.g., *chapatis,* curry, sweets, tea, liquor, etc., to travellers in railway stations, choultries, etc. Sometimes, the seeds are mixed with incense wood, and the victim is exposed to the fumes which produce lethargy. The victim soon falls into a deep sleep and later wakes up to find his belongings lost. (2) Homicide and suicide are very rare. (3) Abortifacient. (4) It is believed to have aphrodisiac properties. (5) Accidental cases occur usually in children by eating the fruits. The seeds and leaves are mixed with tobacco or ganja and smoked in a pipe. (6) A decoction of seeds is sometimes added to liquor or toddy to increase the intoxicating property. (7) It is sometimes used as love philter.

A person suffering from delirium of datura is not criminally responsible for his acts.

Mydriatic Test: A drop of solution to be tested is put into the eyes of a cat. The pupils dilate within half hour if datura is present, due to the presence of atropine.

CANNABIS SATIVA OR INDICA

It is also known as Indian hemp, hashish or marijuana, pop, dope, grass, Mary Jone or bhang. The plant grows all over India, but its cultivation is restricted by law (Plate 24-150). The active principles are contained in its resin. The **principal constituent** of the resin are cannabinol, which has no action; cannabidiol is also inert, but on exposure to heat, it is partly converted to the **very active isomeric tetrahydrocannabinols (THC). THC acts on widely distributed specific receptor regions of brain. All parts of the plant, male or female, contain the active material. It is a CNS stimulant. It is the most widely used illicit drug in the world.**

Common preparations: (1) **Bhang** (*siddhi, sabji*)**:** It is prepared from the dried leaves and fruit shoots. It is used as we use tea to prepare a decoction. It is the mildest and **contains 15% of active principle.** (2) **Majoon: It is a sweet prepared with bhang.** (3) **Ganja:** It is prepared from the **flower tops of the female plant.** It has a rusty-green colour and a characteristic odour. It is mixed and smoked with tobacco in a pipe or *hukka.* **It contains 15 to 25% of the active principle. Ganja also known as marijuana,** is used for smoking in cigarettes, which contain 0.3 to 0.6 g. cannabis and are known as ***reefers.*** (4) **Charas or hashish:** It is the resin (dope or shit) exuding from the leaves and stem of the plants and it **contains 25 to 40% of the active principle.** It is dark green or brown in colour. It is mixed and smoked with tobacco in a pipe or *hukka.*

A concentrate of cannabis is produced by repeated extraction to yield a dark viscous liquid called hashish oil. 1 or 2 drops of oil is added to tobacco of a cigarette and smoked.

Signs and Symptoms: They appear soon after smoking and last for 1 to 2 hours, and within half hour after swallowing and last for two to three hours. Taken

in small dose the effects are very slight, which usually include euphoria, passivity, heightening of subjective experiences, and disorientation. With moderate doses these effects are intensified by impaired immediate memory function, disturbed thought patterns, lapses of attention, and a subjective feeling of unfamiliarity. **High doses produce changes in body image, depersonalisation and marked sensory distortion.**

Symptoms of Intoxication: (A) Psychiatric: (1) Feelings of detachment, cleverness, disinhibition, depersonalisation, euphoria, elation, relaxation, well-being, dreaminess, sleepiness, self-confidence, jocularity, laughing, silliness, rapidly changing emotions. (2) Thought processes: irrelevant thoughts, altered reality testing, decreased concentration and attention span, altered sense of identity, disorientation. (3) Sensory novelty and increased awareness of stimuli: vivid images, illusions and hallucinations. (4) Feelings of precordial distress and tightness in chest; fear of dying. (5) Altered concepts of time and space. Change in body image, self-confidence, altered sexual feelings. (6) Maladaptive behavioural effects: impaired judgement, failure to meet responsibilities. (7) Speech changes: rapid, impaired, talkative, flighty, poor immediate memory. During the so-called "trip" as distortion of perception occurs, the users feel that they could fly from high windows or stop passing buses with their bare hands.

(B) Physical: Increased appetite and thirst, slight nausea, heaviness and pressure in the head, dizziness, somnolence, paraesthesias, restlessness, ataxia, tremors, dry mouth, tachycardia, urinary frequency, injected conjunctivae.

Fatal Dose: Charas 2 g.; ganja 8 g; bhang 10 g/kilo.; THC 30 mg/kg.; body weight.

Fatal Period: Several days.

Treatment: (1) Stomach wash or emesis, activated charcoal and cathartic. (2) 100 ml. of 50% glucose, 2 mg. naloxone, and 100 mg. thiamine i.v. (3) 5 to 10 mg. diazepam, if the patient is aggressive. (4) Assure the patient. (5) If flashbacks occur give antianxiety and if necessary antipsychotic drugs, such as haloperidol. (6) Psychotherapy.

Postmortem Appearances: These are not characteristic except asphyxial signs.

Chronic Poisoning: The use of the drug in small quantities even for long periods is not harmful. **Tolerance and psychological dependence develop.** Used in excess, it causes degeneration of the central nervous system and insanity. There is loss of appetite, weakness, wasting, tremors, vacant look, red eyes, impotence, and moral and mental deterioration. Rarely they become insane and may suffer from hallucinations and delusions of persecution. Heavy marijuana users may develop manic or paranoid psychosis. **The person may run amok, i.e., he develops a psychic disturbance marked by a period of depression, followed by violent attempts to kill people (impulse to murder). He first kills a person against whom he may have real or imaginary enmity and then kills anyone that comes in his way until the homicidal tendency lasts.** Then he may commit suicide or may surrender himself. If the abuse is continued for a considerable time, it may lead to behavioural problems, crime and even mental derangement. Chronic use may lead to **amotivational syndrome** with loss of age appropriate behaviours, like lethargy, lack of interest in day-to-day activities at home and school.

Poisoning: (1) Most of the cases of poisoning are due to overindulgence, but there may be accidental ingestion or inhalation. (2) **Majun and charas are sometimes used by road poisoners to stupefy persons to facilitate robbery.** (3) It is sometimes taken by criminals before committing a criminal act, to strengthen the nerves. (4) It is used as an aphrodisiac and is supposed to increase the duration of coitus. (5) It is usually taken only once in a day in the evening when the person feels tired. *Fakirs* often believe that bhang frees them from worldly attachments and brings about participation with the divine spirit.

As the withdrawal syndrome is mild, supportive and symptomatic treatment is given.

COCAINE

It is obtained from the leaves of Erythroxylum coca, which grows in India, South America, Java, etc. It is a colourless, odourless, crystalline substance with bitter taste. It is used as local anaesthetic. It is also known as coke, snow, crack, cadillac and white lady. **It is rapidly absorbed from the mucous membranes and from the subcutaneous tissues. The usual routes of intake are by application to the nasal mucous membrane (snorting) and by the i.v. route**

(skin popping). It is also smoked. Its action is short, and as such it has to be taken every one-fourth to one hour to maintain a high. It is an excitant and stimulator of autonomic nervous system. **Cocaine, nicotine, amphetamine and barbiturates can be found in the stomach, even when given parenterally. Its action is somewhat like that of amphetamine.**

Signs and Symptoms: (1) **Stage of Excitement:** There is bitter taste, dryness in the mouth, dysphagia, feeling of well-being and loss of depression and fatigue. The patient may be excited, restless and talkative, but this passes into a calm, dull condition. The pulse is rapid, blood pressure falls, respirations rapid and deep, pupils dilated, headache, pallor of the skin, cyanosis, sweating and the temperature is raised. The reflexes are exaggerated and there may be tremors or convulsions. Occasionally, the patients may have hallucinations and become maniacal. There is often a feeling of tingling or numbness in the hands and feet, and a numb feeling at the place where the drug has touched, e.g. nose and back of throat when it has been sniffed. (2) **Stage of Depression:** Within an hour or even less, respirations become feeble, profuse perspiration, collapse, convulsions and death occurs. Sudden death can occur following i.v. injection and smoking. Death is due to respiratory failure, cardiac failure, or vascular collapse. Death may occur rapidly from cardiac arrest in cocaine overdose or hypersensitivity.

Cocaine produces hypertension, which like amphetamine may lead to cerebral bleeding. Large doses or a "binge" may result in anxiety and panic leading to paranoia. A combination of cocaine and heroin taken by injection is known as **"speedball".**

Fatal Dose: One gram orally.

Fatal Period: Few minutes to few hours.

Treatment: (1) Gastric lavage with warm water containing potassium permanganate, charcoal or tannic acid. (2) If applied to the nose or throat, wash out the mucous membrane with water. (3) If injected, apply a ligature above the part. (4) Amyl nitrite is antidote and is given by inhalation.

Postmortem Appearances are those of asphyxia.

Cocaine Habit: It is also known as cocainism, cocainophagia or cocainomania. It causes digestive disturbances, anorexia, salivation, tachycardia, tachypnoea, insomnia and emaciation. The face is pale, eyes sunken, pupils dilated, and the gaze "shifty". The tongue and teeth are black. The sniffing habit leads to ulceration of the nasal septum. Degeneration of central nervous system occurs, and the patient may become violent, aggressive and suffer from hallucinations, convulsions, delirium and insanity. **Magnan's symptom or cocaine bugs is characteristic, in which there is a feeling as if grains of sand are lying under the skin, or some small insects are creeping on the skin, giving rise to itching sensation** (formication; tactile hallucinations).

The Circumstances of Poisoning: (1) It is rarely used for homicide or suicide. (2) Accidental cases occur, due to addiction from internal use, hypodermic injection and from urethral, vesical and rectal injection. (3) It is believed to be an aphrodisiac and to increase the duration of sexual act by paralysing sensory nerves of glans penis. (4) Prostitutes sometimes inject cocaine solution into vagina to produce local constriction.

CHAPTER

32

Drug Dependence and Abuse

Drugs: A drug is any substance, or product that is used or intended to be used to modify or explore physiological systems or pathological states for the benefit of recipient (WHO).

Addiction is defined as a chronic disorder characterised by compulsive use of drugs (craving) resulting in physical, psychological and social harm; and continued use despite evidence of that harm.

Drug addiction is a state of periodic or chronic intoxication, produced by the repeated compulsive consumption of a drug resulting in physical, psychological, and social harm and is harmful to the individual and to the society. The most important drugs of addiction are alcohol, opium and its derivatives, pethidine, heroin, barbiturates, cocaine, cannabis, LSD, benzodiazepines, buprenorphine, dextropropoxyphene, and amphetamine. Other drugs are chloral hydrate and bromides. **Heroin is the most dangerous. Drug habituation** (habit) is a condition resulting from the repeated consumption of a drug, in which there is psychological or emotional dependency on the drug. Caffeine and nicotine are habit-forming drugs. **Drug dependence** includes both the terms 'addiction' and 'habituation'. For drug dependence to exist, psychological dependence must be present but physical dependence may not necessarily occur (Table 32-1).

Physical or physiological dependence is a biological phenomenon which depends on the type, dose and duration of drug use irrespective of personality factors. If the drug is abruptly withdrawn, a withdrawal syndrome will occur in a physically dependent person. The withdrawal symptoms are usually opposite to the effects of the drug itself. **Psychological dependence is a compulsive need for a drug in order to maintain a state of well-being, and it can occur in the absence of physical dependence.** Indiscriminate use of any of these drugs becomes dangerous and produces a gradual mental, physical and moral worsening of the individual and sometimes also sexual perversions or crime. To obtain the money for the drug, the addict often turns to prostitution or crime. The majority of drug victims are neurotic individuals who are mentally unbalanced. Hereditary factors, abnormal mental conditions, frustrations in life, anxiety, chronic tensions, physical inability to do a job, curiosity, etc., are some of the causes of drug addiction.

Tolerance occurs when increased doses are required to produce the same effect and will only

Table (32-1). Difference between drug addiction and drug habituation

Trait	Drug addiction	Drug habituation
(1) Dependence:	Psychological and physical.	Some degree of psychological but not physical.
(2) Compulsion:	Present.	Desire but no compulsion.
(3) Dose:	Tendency to increase.	No tendency to increase.
(4) Withdrawal symptoms:	Characteristic symptoms.	None or mild.
(5) Harm:	Both to the individual and society.	If any, primarily to the individual.

be maintained if the drug is taken regularly and in sufficient doses.

Alcohol and tobacco are the commonest substances abused, followed by sedatives and tranquilisers, cannabis, opiates and cocaine. Amphetamines and hallucinogens are less popular.

In India abused drugs are: alcohol, tobacco, cannabis, opiates, sedatives and tranquilisers. Cocaine and hallucinogens are used less commonly.

Synthetic analogues called designer drugs are manufactured illicitly in clandestine laboratories for recreational use which are highly potent and addictive.

Symptoms of Drug Dependency: Loss of appetite and weight; clumsy movements, unsteady gait, tremors; reddening and puffiness of eyes, unclear vision; slurring of speech; loss of interest, sleeplessness, lethargy and passivity; acute anxiety, depression, profuse sweating, mood changes, temper tantrums; depersonalisation and emotional detachment; impaired memory and concentration; preference for solitude, especially spending long hours in the toilet. Acute intravenous narcotism is characterised by the appearance of fulminant pulmonary oedema and immediate collapse and death. Money and articles disappear from home, and needles, syringes, strange packets, etc. are found at home.

Withdrawal Symptoms: They may begin within 6 to 8 hours following stoppage of the drug or they may be delayed for 24 to 48 hours, depending upon the particular drug being used. The length of period of withdrawal symptoms also varies and can last up to 10 days. The intensity of the symptoms depends on the dose and type of the drug used, the duration of addiction, and the suddenness of withdrawal of the drug. Early symptoms are: chilliness, a sensation of cold, uneasiness, yawning and rhinorrhoea. Later, respirations become laboured, and very rapid. Goose skin, lacrimation, gross tremors and dilatation of pupils are seen. Anorexia is present in all the stages. The third stage is one of sleep lasting from 8 to 16 hours. Upon awakening all the previous symptoms become intense. In addition, there is tachypnoea, fever, hypertension, pain and cramps in the legs and abdomen, perspiration, vomiting and diarrhoea.

Treatment: (1) The person should be removed to an institution. (2) Constant supervision to prevent addict from obtaining secret supplies of the drug. (3) **Detoxification:** This consists of a reduction in dosage of drug over a period of one to three weeks. (4) Administration of drugs, such as sedatives, benzedrine, hyoscine. (5) Diverting the mind by engaging him physically and mentally in some occupation. (6) Psychotherapy (group, family or individual). (7) Improving general health. (8) Symptomatic treatment.

The treatment is successful only in 10 to 25% of cases. **Narcotic addicts may be murdered by a 'hot shot'. This is a dose of narcotic with a poison, such as strychnine in it.** In such cases, only signs of anoxia and cerebral depression are present. Another method of accidental or homicidal death is by the use of a purer drug than the addict has been using.

Cocaine, cannabis, LSD, antidepressants and antipsychotics do not produce physical dependence.

Loss of Tolerance: One of the problems of the addict is loss of tolerance following withdrawal during hospitalisation or imprisonment. The addict on being released often goes back into his old environment and resumes his addiction, taking the same dose that he did before. Such a dose may prove lethal as the addict needs time to build up his tolerance all over again.

Cotton fever: Fever developing due to injection of a water extract of the cotton remaining after the heroin supply is used in a "bag".

Drug abuser's elbow: Myositis ossificans resulting due to repeated needle punctures near the elbow in the intravenous (I.V.) drug abuser.

SOLVENT ABUSE

Volatile substance abuse (solvent abuse, glue sniffing) involves the deliberate inhaling of a variety of substances, such as toluene (glue-sniffing), which is aromatic petrol hydrocarbon, gasoline (petrol), xylene, benzene, methylene and ethylene chloride, fluorocarbons, carbon tetrachloride, butane, propane, etc. for their psychotropic and hallucinogenic properties. Volatiles such as paint stripper, typewriting erasure fluid, brush cleaner, household aerosols, fabric cleaner, gasoline and glue solvent are misused.

Huffing refers to inhaling vapours from a cloth that is saturated with the volatile substance, and held over or near to the nose and mouth. Bagging

refers to inhaling and exhaling into a bag that has been filled with a small amount of a volatile substance. The open end is then placed against nose and mouth and the air inside breathed. Clinical manifestations depend on the substance abused.

Gaseous substances may be introduced directly into the mouth or nose from either a large cylinder or from the small ampoule cylinders for refilling gas cigarette lighters. Others are used directly from pressurised aerosol cans, including pain-relieving sprays. Sniffing is done by inhaling directly from the neck of a container, such as jerry cans and petro-fillers.

The effects vary from a condition resembling alcoholic intoxication and distortion of perception to actual hallucinations. The person feels powerful dreams, heightened sensation and detachment from reality. The sufferer often behaves totally irrationally and may injure or even kill himself. Later the abusers will have complete amnesia for the period of intoxication. Most of the abusers are usually male teenagers. There is no physical withdrawal.

Major cause of death is due to sudden cardiac arrest. Other causes of death are hypoxia and hypercapnoea, plastic bag asphyxia, aspiration of vomit and accidents.

BODY PACKER SYNDROME: The act of swallowing containers, condoms, balloons, plastic bags of packages filled with illegal drugs for the purpose of smuggling has been termed "body-packing". One packet may contain 3 to 7 g. of the narcotic. Drugs such as loperamide or diphenoxylate hydrochloride with atropine may be taken to reduce gut motility and prevent the passage of the packages on a long-distance flight before the end of journey. They can conceal up to ½ kg of drug. On arrival at his destination, the courier takes a laxative, recovers the packets and passes them on to the "pusher" who distributes the drug. Sometimes packets become unsealed or burst in the small intestine, especially cocaine-filled containers, allowing massive absorption and cause the courier's death from poisoning. Even if the packets do not rupture, osmotic seepage across the latex wrapping allows small amounts of drug to appear in the circulation and urine.

BODY STUFFER SYNDROME: Persons arrested swallow illegal drugs for concealing the evidence from authorities. This is termed "body stuffer". Drugs may be concealed in the ears, mouth, nose, vagina or rectum. Most body packers may be diagnosed by X-ray of the abdomen, as the packages are often radio-opaque. All packets are seen with computed tomography (CT) scanning or barium contrast studies.

Treatment: (1) Diazepam 10 mg i.v. followed by 5 mg. i.v. every 5 min. until the patient becomes calm. (2) Give glucose, thiamine and multivitamins. (3) Fluid and electrolyte balance. (4) Oxygen.

GLOSSARY

Acid head = Heavy user of LSD.

Bad or bum or freak-out trip = An LSD experience in which the drug effects are unpleasant and sometimes frightening. It usually lasts from 8 to 12 hours.

Busted = To be arrested.

Cutting = Mixing a drug, usually a narcotic, with other substances.

Flip = To go psychotic.

Flashback = A transitory, spontaneous recurrence of drug-induced experience in a drug-free state.

Guide = A person who "babysits" for the psychedelic user during a session.

Hangover = Temporary illness usually following recovery from drunkenness.

Mainlining = I.V. injection.

Physical dependence = Physiologic requirement for a drug to prevent symptoms of withdrawal.

Shooting = I.V. injection.

Pot = Marihuana.

Psychological dependence = The mind's need to continue taking drug.

Return trip = Reappearance of LSD-like effects long after the last dose of LSD was taken.

Skin popping (joy popping) = I.M. injection.

Trip = effects of LSD.

Psychotomimetic = Psychosis mimicking.

Soft drugs = Amphetamine, barbiturates, cannabis, LSD.

Hard drugs = Opium, heroin, cocaine, methedrine.

CHAPTER

33 Spinal Poisons

STRYCHNOS NUX VOMICA

Strychnine *(kuchila)* is a powerful alkaloid obtained from the seeds of Strychnos nux vomica, which are found in the jungles in India. **The seeds of nux vomica contained in the ripe fruit are poisonous.**

Active principles: The seeds contain strychnine and brucine up to one-and-half percent of each. The seeds also contain a glucoside, loganin. It is intensely bitter. **All parts of the tree are toxic.** Brucine is allied to strychnine in composition and action. Strychnine is 10 to 20 times more poisonous than brucine.

Seeds: The seeds are flat, circular discs, or slightly convex on one side, concave on the other, two-and-half cm in diameter, six mm in thickness. They are ash-grey or light-brown in colour, have a shining surface and covered with radiating silky fibres (**Plate 23-139**). They are very hard, tough and difficult to be reduced to fine particles. The bark, wood and leaves contain brucine but no strychnine. Strychnine is used as a respiratory stimulant, as a rodenticide and for killing stray dogs.

Absorption and Excretion: All mucous membranes absorb strychnine. Much is taken up by the liver and muscles. The release of strychnine from the liver and muscles produces convulsions on second or third day of poisoning, after sedation is discontinued. About 80% is oxidised mainly in the liver. It is excreted slowly by the kidneys and traces in bile, milk and saliva. It may be found in the cadaver up to 4 years.

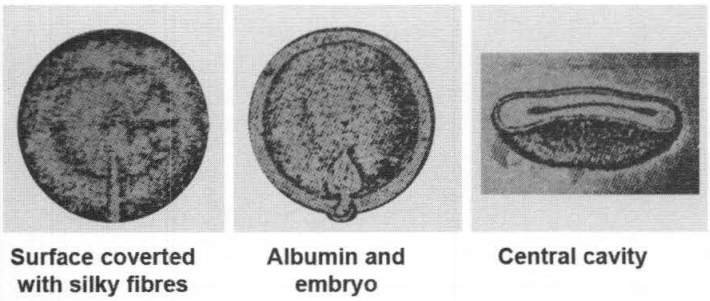


Fig. (33-1). Nux vomica seeds.

Action: It depresses the inhibitory postsynaptic potentials in the spinal cord and prevents the effects of glycine (the presumed inhibitory transmitter). **Widespread inhibition in the spinal cord results in "release" excitation. The action is particularly noted in the anterior horn cells.** It stimulates the cerebral cortex. GABA, the neurotransmitter for presynaptic inhibitory neurons is not affected.

Signs and Symptoms: If swallowed uncrushed, the seeds of nux vomica have no poisonous action, as they are not dissolved in the gastrointestinal tract, and are passed entire in the faeces. When crushed seeds are taken, the symptoms are delayed for an hour or more. If the alkaloid is swallowed, the symptoms occur within 5 to 15 minutes. Bitter taste in the mouth, sense of uneasiness and restlessness, feeling of suffocation and great fear and difficulty in swallowing occur. The convulsions are preceded by such prodromal symptoms as increased acuity of perception, increased rigidity of muscles and muscular twitchings. Convulsions are produced due to direct action on the reflex centres of spinal cord and affect all the muscles at a time. These are at first clonic, but eventually become tonic. During the convulsions, the face is cyanosed and has anxious look, eyes are staring, eyeballs prominent and pupils are dilated. Risus sardonicus results from contraction of the jaws and facial muscles in which the corners of the mouth are drawn back. The mouth is covered with froth, frequently bloodstained. **The convulsions are most marked in antigravity muscles, so that the body typically arches in hyperextension (opisthotonus).** In supine position, the body is supported by the heels and head. The head is bent backwards, and the whole of the body becomes rigid, often assuming a bow-like form. Sometimes, the spasm of the abdominal muscles

Table (33-1). Difference between strychnine poisoning and tetanus

	Trait	Strychnine	Tetanus
(1)	History:	No history of injury.	History of injury present.
(2)	Onset:	Sudden.	Gradual.
(3)	Convulsions:	All muscles of the body are affected at a time.	All muscles are not affected at a time.
(4)	Lower jaw:	Does not start in, nor especially affect the jaw.	Usually starts in and especially affects lower jaws.
(5)	Muscular condition:	Between fits muscles are completely relaxed.	Between fits muscles are slightly rigid.
(6)	Fatal period:	One to two hours.	More than twenty-four hours.
(7)	Chemical analysis:	Strychnine found.	No poison found.

may bend the body forward (**emprosthotonus**), or to the side (**pleurosthotonus**). Consciousness is not lost and the mind remains clear till death. The duration of convulsion varies from half to two minutes. In between the convulsions, the muscles are completely relaxed and the patient looks well though somewhat exhausted, and the breathing is resumed (Table 33-1). After 5 to 15 minutes or on slightest impulse, e.g., a sudden noise, a current of air or gently touching the patient, another convulsion occurs. In fatal cases, the convulsions rapidly succeed one another, and increase in severity and in duration, and death usually occurs after 4 to 5 convulsions. **The patient cannot breathe because the diaphragm and thoracic muscles are fully contracted.** Hypoxia causes medullary paralysis and death.

Fatal Dose: 50 to 100 mg; one crushed seed.

Fatal Period: One to two hours.

Treatment: (1) The first step is the effective control of convulsions, i.e., the symptoms treated before the disease. The patient should be kept in bed in a dark room, free from noise and disturbance. Convulsions may be controlled initially with diazepam, one-tenth to half mg. kg. i.v. slowly, and then phenobarbital i.v. If these prove ineffective, ether may be administered to the point of unconsciousness. (2) **Short-acting barbiturates like pentobarbital sodium, or sodium amytal are antidotes to strychnine and should be given in a dose of 0.3 to 0.6 g. i.v.** (3) Wash the stomach with warm water and dilute solution of potassium permanganate and then introduce a suspension of activated charcoal to adsorb strychnine, which should be removed later. Tannic acid may be used if charcoal is not available.

Postmortem Appearances: There may be signs of asphyxia.

Physiological Test: Injection of an aqueous solution of the suspected material into the dorsal lymph sac of a frog, will produce tetanic convulsions in a few minutes if strychnine is present. Later, stimulation of the frog will produce convulsions.

The Circumstances of Poisoning: (1) Suicide and homicide are rare. (2) Cattle poison. (3) Arrow poison. (4) Aphrodisiac.

CHAPTER

34 Cardiac Poisons

NICOTIANA TABACUM (Tobacco)

All parts are poisonous except the ripe seeds. The dried leaves *(tambaku)* contain 1 to 8% of nicotine and are used in the form of smoke or snuff or chewed. The leaves contain active principles, which are the toxic alkaloids nicotine and anabasine (which are equally toxic), and nornicotine (less toxic). Nicotine content in a cigarette ranges from 15 to 20 mg. Smoking usually results in absorption of 1 to 2 mg. per cigarette.

ABSORPTION AND EXCRETION: Nicotine is rapidly absorbed from all mucous membranes, lungs and the skin. It is slowly detoxicated in the liver. It is excreted by the kidneys.

ACTION: It acts on the autonomic ganglia which are stimulated initially, but are depressed and blocked at later stage.

CHRONIC POISONING: Symptoms are cough, wheezing, dyspnoea, anorexia, vomiting, diarrhoea, anaemia, faintness, tremors, impaired memory, amblyopia, blindness, irregularity of the heart with extrasystoles and occasionally attacks of pain suggesting angina pectoris.

Nicotine replacement therapy (NRT) includes use of nicotine products including gum, transdermal patch, nasal spray, lozenge and inhaler. Bupropion can be used in those who are motivated to quit. Clonidine and nortryptyline can be used as second line of treatment.

FATAL DOSE: 60 to 100 mg. of nicotine; 15 to 30 g. of crude tobacco.

FATAL PERIOD: 5 to 15 minutes.

TREATMENT: (1) Wash the stomach with warm water containing charcoal, tannin or potassium permanganate. (2) A purge and colonic wash. (3) Symptomatic.

POSTMORTEM APPEARANCES: They are those of asphyxia.

For malingering, tobacco leaves are soaked in water for some hours and placed in axillae at bedtime, which is held in position by a bandage. Poisonous symptoms are seen the next morning.

DIGITALIS PURPUREA

Entire plant is toxic, containing over thirty cardiac and steroidal glucosides. The root, leaves and seeds of digitalis contain digitoxin, digitalein, digitonin and digoxin (glycosides).

SIGNS AND SYMPTOMS: There is nausea, vomiting, abdominal pain, diarrhoea, faintness, dizziness, oppression in the chest, disorientation, nervous irritability, tingling and coldness in extremities, depressed and then accelerated heart rate, varying degrees of atrioventricular block, ectopic beats, coupled rhythm, atrial and ventricular fibrillation. The patient becomes drowsy, coma sets in and death occurs from cardiovascular collapse.

FATAL DOSE: 15 to 30 mg. of digitalein: 4 mg of digitoxin; leaf, 2 g.

FATAL PERIOD: 1 to 24 hours.

TREATMENT: (1) Stomach wash with a solution of tannic acid. (2) The bowels should be evacuated. (3) Digoxin-specific antibody fragments (Fab), 0.4 to 0.8 g. i.v. in half an hour.

POSTMORETEM APPEARANCES: They are not characteristic.

NERIUM ODORUM

Nerium odorum (white oleander; *kaner)* grows wild in India. Flowers are white, pink, dark-red or rarely pale-yellow (**Plate 24-145**).

Active principles: All parts of the plant are poisonous, containing several cardiac glycosides, primarily **oleandroside (oleandrin), and nerioside (nerin)**, which resemble digitalis in action. It also contains **folinerin and rosagenin**. The nector yields poisonous honey.

Signs and Symptoms: Ingestion causes difficulty in swallowing and articulation, abdominal pain, vomiting, profuse frothy salivation and diarrhoea. Pulse is first slow and later rapid and weak, blood pressure falls, fibrillation, AV block, respirations are increased, pupils are dilated, muscular twitchings, tetanic spasms, lockjaw, drowsiness, coma, respiratory paralysis and death usually occurs from cardiac failure.

Fatal Dose: 15 to 20 g. of the root; 5 to 15 leaves.

Fatal Period: 24 to 36 hours.

Treatment: Wash out the stomach and treat the symptoms.

Postmortem Appearances: They are not characteristic. Congestion of organs is seen.

The Circumstances of Poisoning: (1) The root, leaves or fruit are often used as a paste or decoction for suicidal purposes. (2) Homicide is rare. (3) As an abortifacient, root is used either locally or taken internally. (4) Root is taken internally for treating venereal diseases. (5) Root is used for treating cancers and ulcers in the form of paste. (6) The decoction of leaves is applied externally to reduce swelling. (7) As a cattle poison, the juice of root is applied on piece of cloth and inserted into the anus of the animal. (8) Smoke from the burning plant is toxic. **When plant material is used to roast food over a fire, the poisonous sap transferred to the food may be lethal.**

CERBERA THEVETIA

All parts of cerbera thevetia (yellow oleander, *pila kaner*) are poisonous. The flowers are large, bell-shaped and yellow (**Plates 23-141 and 24-146**).

Active principles: The seeds contain cardiac glycoside **thevetin, and thevetoxin**, similar to digitalis in action; **nerifolin, peruvoside, and ruvoside, cerberin** and also a bitter principle that acts on the CNS and produces tetanoid convulsions. Milky juice exudes from all parts of the plant.

Signs and Symptoms: Chewing the bark or seed kernel causes a slight numbing sensations and feeling of heat in the mouth and purging. Ingestion causes burning pain in the mouth, dryness of throat, tingling and numbness of tongue, vomiting, diarrhoea, headache, giddiness, dilated pupils, loss of muscular power and fainting. Pulse is rapid, weak and irregular, blood pressure is low. Heart block, collapse and death from peripheral circulatory failure occurs.

Fatal Dose: 8 to 10 seeds; 15 to 20 g. of root. 5 to 10 leaves.

Fatal Period: 2 to 3 hours.

Treatment: (1) Wash out the stomach. (2) Sodium molar lactate transfusion with glucose and one mg. atropine, 2 ml. adrenaline and 2 mg. noradrenaline is beneficial. (3) Symptomatic.

Postmortem Appearances: They are not specific.

The Circumstances of Poisoning: (1) The root and seeds are used sometimes for suicide or homicide. (2) Root and seeds are taken for criminal abortion. (3) For cattle poisoning, the seeds are crushed and fed to the animal with corn or bread.

Fig. (34-1). Cerbera odollam.

CERBERA ODOLLAM *(pilikirbir)*

This plant is closely allied botanically to cerbera thevetia. The flowers are white, like those of jasmine. Milky acrid juice exudes from all parts of the plant (**Fig. 34.1**). The active principles are **cerberin, cerberoside, odollin, odollotoxin, thevetin and cerapain.**

Signs and Symptoms: They appear within one hour. The initial symptoms are gastrointestinal. There is bitter taste, nausea, severe retching, vomiting, abdominal pain and in few cases diarrhoea, general weakness, blurring of vision, sinus bradycardia, irregular respiration, collapse and death from heart failure.

Fatal Dose: Kernel of one fruit.

Fatal Period: 1 to 2 days or more.

Postmortem Appearances: They are those of asphyxia.

Treatment: (1) Stomach wash. (2) Atropine half mg. i.v. and repeated every 15 to 30 minutes to keep heart rate above 50 per minute.

Circumstances of Poisoning: (1) For suicide, the kernels are taken as such, or after grinding it with palmyra jaggery or molasses or by preparing a curry with it. (2) For homicide, the powdered kernel is added to alcohol. (3) Bark, leaves and milky juice are used as emetic and as purgative.

ACONITE

There are several varieties of aconite (monk's hood, blue rocket, *mitha zaher, bish, bikh*), but the roots of Aconitum napellus and Aconitum ferox are commonly used. It grows in the Himalayas. All

PLATE 21

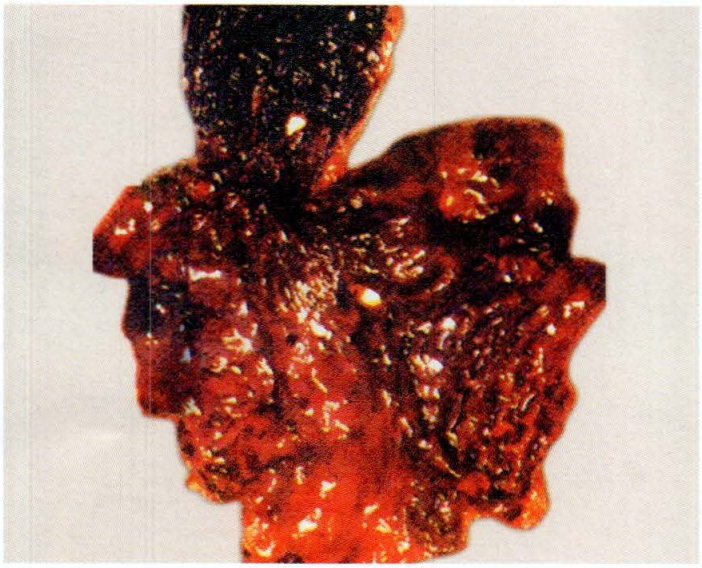


Fig. 121. Stomach in caustic soda poisoning.

Fig. 122. Stomach in aspirin poisoning.

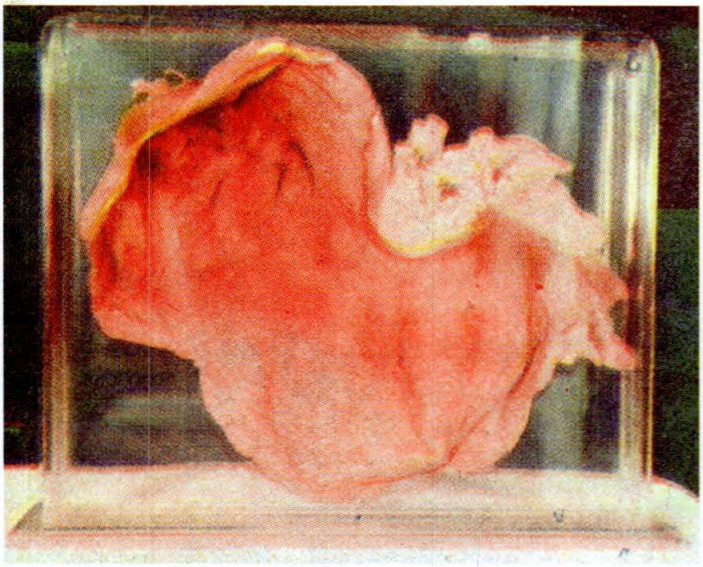


Fig. 123. Stomach in hydrocyanic acid poisoning.

Fig. 124. Stomach in sodium cyanide poisoning.

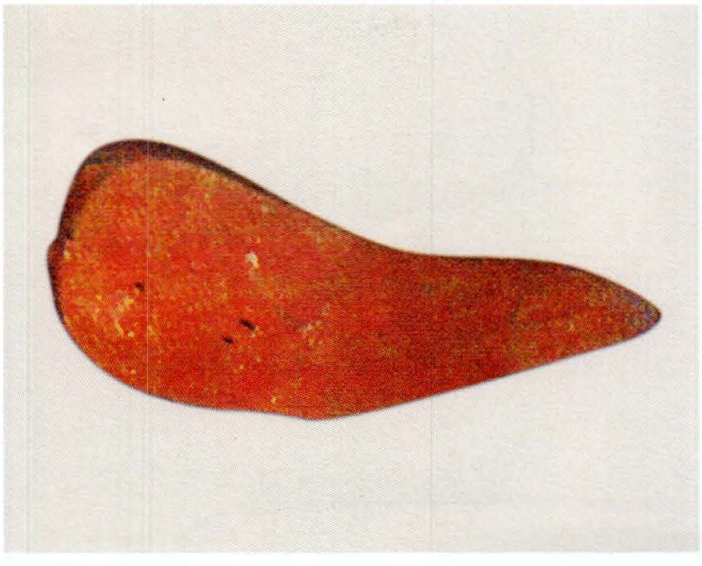


Fig. 125. Liver in hydrocyanic acid poisoning.

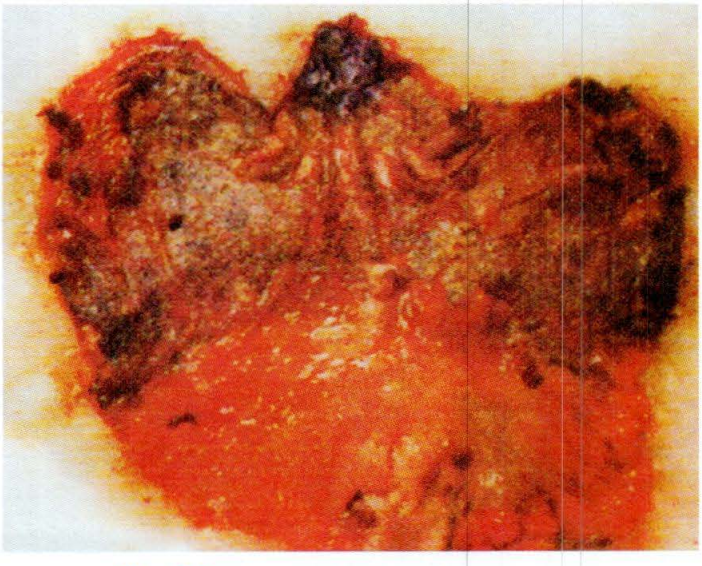


Fig. 126. Stomach in lysol poisoning.

PLATE 22

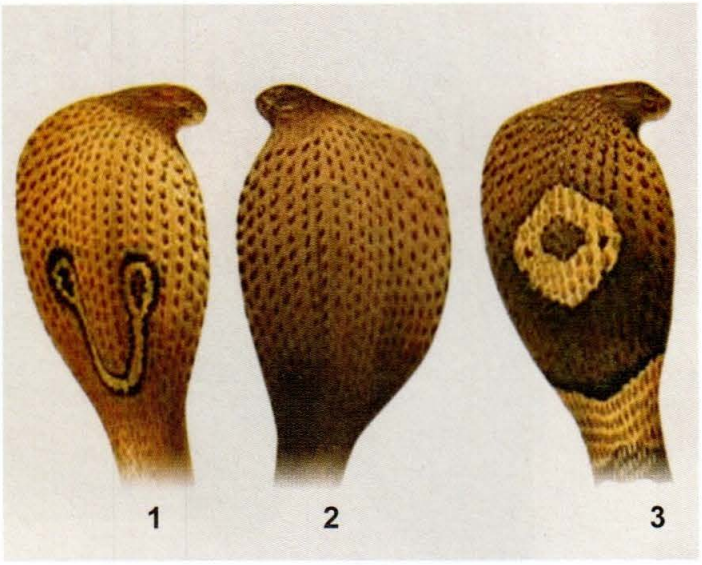


Fig. 127. Cobra: (1) Binocellate, (2) Black, (3) Monocellate.

Fig. 128. Common krait *(Bungarus caeruleus).*

Fig. 129. Banded krait.

Fig. 130. Saw-scaled viper.

Fig. 131. Russell's viper.

Fig. 132. Lizard: Northern house gecko.

Fig. 133. Centipede.

PLATE 23

Fig. 134. Ricinus communis.

Fig. 135. Croton tiglium.

Fig. 136. Abrus precatorius.

Fig. 137. Datura.

Fig. 138. Capsicum annum.

Fig. 139. Nux vomica.

Fig. 140. Semecarpus.

Fig. 141. Cerbera thevetia.

Fig. 142. Poppy.

(Poisonous Seeds)

PLATE 24

Fig. 143. Calotropis gigantea.

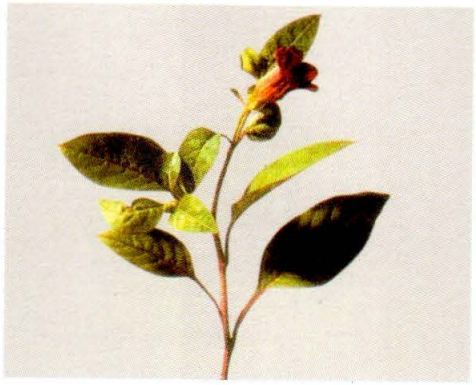


Fig. 144. Belladonna plant.

Fig. 145. Nerium odorum.

Fig. 146. Cerbera thevetia.

Fig. 147. Ricinus communis.

Fig. 148. Datura alba.

Fig. 149. Argemone mexicana.

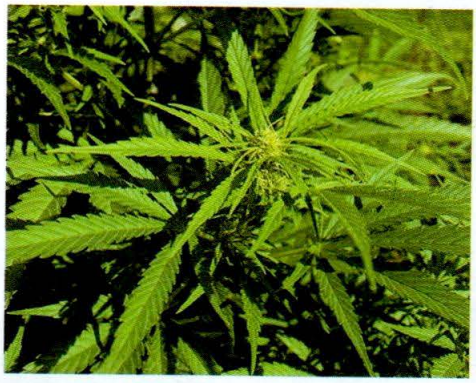


Fig. 150. Cannabis sativa.

varieties and all parts of the plant are poisonous. The root is most potent, contains aconitine and ten or more other alkaloids, such as pseudo-aconitine, indaconitine, bikhaconitine, picraconitine, aconine, mesaconitine, jesaconitine, etc. Aconitine stimulates and then depresses CNS.

Dry root: The dry root is conical or tapering and shows scars or bases of broken rootlets and is arched or shrivelled with longitudinal wrinkles. It is usually 5 to 10 cm. long and 2 cm. thick at the upper end. The external colour is dark-brown and when freshly cut the internal colour is white, which becomes pink on exposure to air. It is odourless but the taste is sweet, then acrid. When dried and soaked in oil the root is black, heavy, hard and brittle with a strong offensive odour (**Fig. 34-2**). The root is mistaken for horseradish root. Aconite stimulates and then depresses CNS.

Signs and Symptoms: Leaves handled or rubbed on the skin, produce tingling and numbness; so also the root if held for a long time in the hand. The odour of the plant has a narcotic effect. The pollen causes pain and swelling in the eyes. The symptoms occur immediately or within a few minutes. There is a burning sensation from the mouth to the stomach, and tingling and numbness in the mouth, tongue and pharynx. This is followed by salivation, nausea, vomiting and diarrhoea. Later the mouth is dry, and patient suffers from thirst and is unable to swallow. Tingling and numbness are then felt all over the body. There is headache, giddiness, pallor, profuse sweating, subnormal temperature, the limbs become weak and the patient is unable to walk or stand. There may be twitching of the muscles with darting pains, and cramps and convulsions may occur. **The pupils alternately contract and dilate (hippus), but remain dilated in the later stages.** The vision

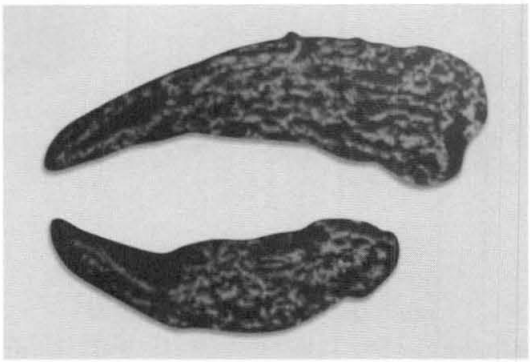


Fig. (34-2). Dried roots of *Aconitum napellus*.

becomes dim and there may be diplopia. Hypotension, cardiac arrhythmia with AV block occurs. At first there is tachycardia, but in later stages bradycardia occurs due to AV block. The mind usually remains clear, although there may be hallucinations. There is marked general muscular weakness, oppression in the chest, and death occurs from paralysis of heart or respiratory centres or both.

Fatal Dose: 1 g. root; 2 to 5 mg. of aconitine.

Fatal Period: 2 to 6 hours.

Treatment: (1) Gastric lavage with warm water and weak solution of iodine in potassium iodide or tannic acid to precipitate alkaloid, or animal charcoal. (2) Atropine half to one mg. is useful. (3) Multiple dose activated charcoal. (4) Symptomatic.

It is eliminated by all routes, but mainly in the urine.

Postmortem Appearances: They are not characteristic but are those of asphyxia.

The Circumstances of Poisoning: (1) Accidental poisoning is due to eating the roots in mistake for horseradish root, or from taking liquor to which aconite is added to increase intoxication. (2) Suicidal cases often occur. (3) It is given with betel leaf to disguise its taste for homicide. (4) Root is used as an abortifacient. (5) Cattle poison. (6) Arrow poison.

CHAPTER

35 Asphyxiants

CARBON MONOXIDE

It is a colourless, odourless, tasteless, nonirritative gas, which is produced due to incomplete combustion of carbon. It burns with a blue flame.

Action: CO is readily absorbed across the alveolus and combines with haemoglobin. Normal blood contains 21% oxygen, with 18 to 19% bound to haemoglobin and 2% dissolved in plasma. About 10 to 20% CO is present in extracellular tissues combined with myoglobin and haemoproteins. **CO affinity to myoglobin is about 40 times greater than oxygen, which may cause direct myocardial depression. CO has 200 to 300 times greater affinity for haemoglobin compared to oxygen. It displaces oxygen from its combination with haemoglobin and forms a relatively stable compound known as carboxyhaemoglobin.** CO is a potent cellular toxin. It effectively and firmly binds to haemoglobin and myoglobin. It inhibits the electron transport by blocking cytochrome A_3 oxidase and cytochrome P-450, and therefore intracellular respiration. **It acts as a chemical asphyxiant by reducing the oxygen carrying capacity of the blood and produces death due to anaemic anoxia.** CO is not absorbed by a body after death.

Elimination: It is eliminated through lungs; about 1% is metabolised to CO_2. Half-life of CO in a healthy adult breathing 21% of oxygen (room air) is 4 to 5 hours. Breathing 100% O_2, it is reduced to 80 to 90 minutes. Hyperbaric oxygen (3 atmospheres pressure) reduces half-life to less than 30 minutes.

Sources: The common sources of CO include coal gas, smoke from fire and the fumes from defective heating appliances, e.g. furnace, stove, water heater, burning oil lamps. It is also found as a component of the fumes of coke kilns, lime kilns, explosion in mines, detonation of explosives. The exhaust gas of motor cars contain 1 to 7 percent of CO, which makes the air of small garage poisonous in 5 to 10 minutes. Diesel engines produce far less CO than petrol engines. Industry accounts for about 20%. In a person smoking one pack of cigarettes per day CO levels are about 5 to 6% and in a heavy smoker 15 to 20%. Traffic policemen may have up to 10% saturation. In haemolytic anaemia, COHb levels may reach 8%.

Signs and Symptoms: Symptoms are stealthy and progressive so that victims may not notice anything, except a headache until they pass into coma. **Symptoms are often mistaken for symptoms of influenza or illness caused by eating contaminated food. The effects of CO are simply those of suboxia.** The sequelae of damage to the CNS include monoplegia or hemiplegia. Impairment of higher intellectual functions, personality changes, cerebellar damage and severe Parkinsonism may occur and may be delayed by several weeks after apparent recovery. **Bullae tend to be discrete and isolated lesions.** The bullous fluid is usually thick and cellular and there is often an inflammatory reaction in the surrounding skin. These bullae are localised by external pressure. These are caused due to skin hypoxia. They rarely involve fingers and toes. As most deaths are relatively rapid, blisters are rare. **There is a tendency of the dying victim to wild, swinging, erratic movements inside the room disturbing clothing and furniture, which gives an impression of violent struggle** (CO automatism). The saturation required to cause death varies with the age and health of the person. Death usually occurs when 80% of haemoglobin is saturated with CO. Senility or the coexistence of any respiratory or circulatory deficiency, or of anaemia or in association with depressive drugs like barbiturates, result in a significant reduction of maximum lethal saturation, and death can occur from as little as 30% saturation. Foetus is particularly sensitive to CO.

Persons with long-term survival following significant exposure **may have a Parkinsonian syndrome or may develop even worse neurological states.**

The Effect of Different Air Concentrations of CO: The upper limit of safety is 0.01% CO in air. If a person breathes CO in low concentration for a considerable length of time, especially during sleep, he will be poisoned just as effectively as though he were exposed to a high concentration for a shorter period. When low concentrations of gas are inhaled, coma does not supervene immediately, but **muscular weakness occurs and a state of complete helplessness may develop and the victim may not be able to save himself.** A concentration of 0.5 to 1% of CO in the atmosphere can produce COHb saturation levels of 75% in 2 to 15 minutes. Exposure to atmosphere containing 0.2% of gas will cause death in about 4 hours, 0.4% in one hour, and 10% in half an hour (Table 35-1).

Treatment: (1) **Remove the patient to fresh air immediately.** If he is conscious and breathing, no treatment is required. (2) Any patient with COHb level greater than 25% should be treated. The most widely accepted treatment is **100% oxygen provided by tight-fitting mask preferably with rubber seals, and endotracheal intubation** for patients with depressed mental status. (3) CO_2 should not be given. (4) **Hyperbaric oxygen (HBO) therapy has several disadvantages,** such as vomiting, convulsions, agitation, rupture of tympanic membranes, blocked sinuses, etc. (5) Gastric lavage early in the course of treatment may prevent aspiration pneumonitis. (6) A whole blood transfusion is useful. (7) The patient should be kept at complete rest for at least 48 hours. (6) Avoid stimulant drugs. (8) Give antibiotics as a prophylaxis against lung infection.

Postmortem Appearances: A **cherry-red colouration of the skin**, mucous membranes, conjunctivae, nail beds, areas of hypostasis, blood, tissues and internal organs **is seen in 15 to 20% of cases only. It is associated with 30 to 40% COHb.** The cherry-red discolouration changes to dark-green, then to brown with the onset of decomposition. The blood is fluid, hyperaemia is general, and serous effusions are common. Fine froth may be seen at the mouth and nose. **Muscle necrosis and blebs of the skin are common.** Congestion of the lungs with pink fluid blood and if the victim survived for some time, pulmonary oedema with congestion are found. The lungs may show bronchopneumonic consolidation. Necrobiosis of the heart muscle and pleural and pericardial anoxial haemorrhages are common. **Bilateral symmetrical necrosis of the lenticular nuclei and punctiform haemorrhages in the white matter of the brain with widespread oedema are common.** Haemorrhages in the meninges and cortex, and selective cellular necrobiosis of ganglion cells in the cortex may be seen.

CO persists for many weeks after death, and may be detected even after putrefaction or

Table (35-1). Symptoms of CO poisoning

COHb%	Symptoms
0 to 10%	No appreciable symptoms.
10 to 20%	Breathlessness on moderate exertion, mild headache.
20 to 30%	Headache, irritability, emotional instability, disturbed judgement, defective memory and rapid fatigue.
30 to 40%	Severe headache, nausea, vomiting, dizziness, dimness of vision, confusion, cherry-red discolouration of skin.
40 to 50%	Increasing confusion, sometimes hallucinations, severe ataxia, rapid respirations and collapse with attempts at exertion. Symptoms resemble alcoholic intoxication.
50 to 60%	Syncope or coma with intermittent convulsions, rapid respirations, tachycardia with a weak pulse and a pink or red discolouration of the skin.
60 to 70%	Increasing depth of coma with incontinence of urine and faeces.
70 to 80%	Deep coma with depressed or absent reflexes, a weak thready pulse, shallow and irregular respirations.
Above 80%	Rapid death from respiratory arrest.

embalming or prolonged burial. The cherry-red colour of viscera will persist even in embalmed body or tissues placed in formalin.

Chronic poisoning: Symptoms include headache, confusion, weakness, paraesthesias, visual disturbances, hypertension, hyperthermia, palpitations, atrial fibrillation, bundle branch blocks, decreased cognitive ability, mental retardation, psychosis and Parkinsonism.

The Circumstances of Poisoning: Suicide: In India, suicide by CO is rare.

Accident: Accidents may occur in connection with incomplete combustion of wood, charcoal or coal in ill-ventilated room. Accidental deaths may be caused by use of flueless braziers, exposure to exhaust gas of petrol engines, exposure to gas in mines following underground fires or explosions, and exposure to gas during fires.

Homicide: It is not common.

CARBON DIOXIDE

It is a heavy, colourless gas with a faintly sweet odour. Atmospheric air contains 0.033% CO_2. Carbon dioxide is heavier than air. Common places which may contain CO_2 in excess include manholes, wells, silos and occasionally cellars.

ACTION: The gas is not toxic, but acts as a simple asphyxiant by preventing the tissues from obtaining oxygen.

SIGNS AND SYMPTOMS: Air containing 3% of pure CO_2 does not produce any noticeable effects for some time; above 10% produces ataxia and unconsciousness. With 40% there is dyspnoea, discomfort, and muscular weakness. With 50% there is dyspnoea, a feeling of tightness in the chest, fullness in the head, ringing in the ears, loss of muscular power, followed by drowsiness, unconsciousness and rapid death.

TREATMENT: Artificial respiration and oxygen should be given freely.

POSTMORTEM APPEARANCES: There is marked cyanosis, congestion, suffusion of the eyes, dilation of the pupils and petechial haemorrhages.

Poisoning is usually accidental.

HYDROGEN SULPHIDE (H_2S)

It is colourless, heavy, flammable gas with a smell of rotten eggs. It is formed during decomposition of organic substances containing sulphur. It is often found in large quantities in sewers (sewer gas), cess pools, privy vaults and tannery vats. Poisoning by this gas is almost always accidental, especially in sewer workers. Hydrogen sulphide in combination with methane, nitrogen, hydrocarbons, ammonia and CO_2 formed in sewers is known as sewer gas. It is heavier than air, so it gets accumulated in low lying areas and closed spaces. Normal tolerance limit is 20 ppm and levels more than 100 ppm is dangerous to life. It interferes with cellular respiration by inhibiting the action of cytochrome oxidase.

SIGNS AND SYMPTOMS: In great dilution there is feeling of dullness and sleepiness, and death may occur during sleep without the victim regaining consciousness. In weak concentration, there is cough, giddiness, nausea and feeling of oppression. The breathing is laboured and heart irregular, cyanosis of the face, inflammation of conjunctivae, lachrymation and photophobia, muscular weakness and prostration. There may be delirium, convulsions or coma and death occurs from asphyxia. If breathed in concentrated form, death occurs immediately from paralysis of respiratory centre. Its toxicity and rapidity of action are comparable to hydrocyanic acid.

TREATMENT: (1) The patient must be removed into the fresh air, artificial respiration and oxygen given. (2) Amyl nitrite inhalation and sodium nitrite infusion in the same dose as for cyanide will hasten the formation of sulphmethaemoglobin.

POSTMORTEM APPEARANCES: Rotten egg odour is given off. The general signs of asphyxia are present. The colour of blood and viscera is greenish-purple.

Nitrous oxide is known as laughing gas.

METHYL ISOCYANATE (MIC)

It is one of the deadliest chemicals and can kill in very small doses when ingested, inhaled or when absorbed through the skin.

SYMPTOMS: Acute irritation of the eyes, lachrimation, blurring of vision, severe burning in the throat, chest pain and laboured breathing, pulmonary oedema. Victims who survive for 5 to 6 days experience the above symptoms and also fever, cough with expectoration often bloody, oedema leading to death. About 10% of the victims have psychiatric symptoms, such as anxiety, depression, sleep disturbances, gas phobia, and a feeling of helplessness. The systemic effects are: liver damage, kidney damage, methaemoglobinaemia and neurological deficits. The isocyanates affect the nucleic acids and thus may be potential teratogenic or mutagenic agents. In females, excessive vaginal discharge, anaemia, stillbirth and foetal abnormalities are seen.

Treatment is purely symptomatic. Sodium thiosulphate is beneficial.

WAR GASES

The term "war gases" includes any chemical (gaseous, liquid or solid), which is used to produce destruction or damage mostly in times of war.

(1) Vesicant or Blistering Gases: These are mainly mustard gas and lewisite, which are volatile liquids. They are discharged in artillery shells so as to impregnate the area of attack. Mustard gas causes irritation of the eyes, nose, throat and respiratory passages, nausea, vomiting and abdominal pain. It passes through the clothes into the skin and produces intense itching, redness, vesication, and ulceration especially of the moist areas.

(2) Asphyxiants or Lung Irritants: These are chlorine and phosgene which are gases, and can be released from tanks, and gas shells. Chloropicrin and diphosgene are liquids which are used in gas shells. Phosgene is 10 times and chloropicrin 4 times more toxic than chlorine. Their action is mainly on the pulmonary alveoli. When inhaled they cause watering of the eyes, coughing, dyspnoea, tightness of chest, headache, vomiting, restlessness, stertorous breathing, cyanosis and collapse. Death occurs in 1 to 2 days due to acute pulmonary oedema.

(3) Lachrymators or Tear Gases: These are mainly chloracetophenone (C.A.P.) which is solid and ethyl iodoacetate (K.S.K.) and bromobenzyl cyanide (B.B.C.) which are liquids. They are fired in artillery shells or penguns. The vapours cause intense irritation of the eyes with a copious flow of tears, spasm of the eyelids and temporary blindness.

(4) Sternutators or Nasal Irritants: These are solid, organic compounds of arsenic and are fired in artillery shells. They are diphenylchlorarsine (D.A), diphenylaminechlorarsine (D.M.), and diphenylcyanarsine (C.D). The vapours cause intense pain and irritation in the nose and sinuses, sneezing, headache, salivation, nausea, vomiting, tightness in the chest and prostration.

The above are used to control riots.

(5) Paralysants: These are hydrocyanic acid, sulphuretted hydrogen and CO.

(6) Nerve Gases: The term nerve gases is incorrect. **The nerve agents are esters of phosphoric acid (liquid) and are identical in their biological activity to organophosphates.** The major agents are GA (tabun), GB (sarin), GD (soman) and VX. The vapours are heavier than air so they tend to sink into valleys, trenches and basements. They are compounds related to phosphate esters in action and toxicity. These are colourless and odourless volatile liquids. They are absorbed from the lungs, gastrointestinal canal, skin or conjunctivae. They inhibit acetylcholine esterase. Exposure to a large amount of vapour will cause loss of consciousness within seconds followed several minutes later by convulsions. Muscles become flaccid and breathing stops. Treatment is similar to organophosphates.

CHEMICAL WARFARE: The offensive use of non-living toxic products produced by living organisms, e.g. toxins, such as botulinum toxin, ricin or saxitoxin is considered chemical warfare. The agents are chemical substances used to injure or kill enemies during war. They produce physiological changes on the human body. Some substances are lethal, and some injure or incapacitate people. These agents are dispersed as tiny droplets through chemical shells, spray tanks, bombs and missiles. Harmful effects are caused when the chemical is inhaled, ingested or when it comes in contact with skin or mucous membrane.

Chemical crowd control agents: The three most commonly used agents are: (1) Orthochlorobenzylidene malanonitrite (CS; teargas), used by law-enforcement agencies and military. (2) 2-chloroacetophenone (CN) (MACE). (3) Oleoresin capsicum (OC), or a combination of these ingredients. They cause acute ocular irritation, lachrymation, conjunctivitis, blepharospasm and extreme discomfort. Effects begin within seconds after exposure and usually lost in 10 to 15 minutes. These agents are available in aerosols or fumes and in particulate form with dispersal device. They are also available in grenades or canisters.

Improvement is usually rapid, and people rarely seek medical attention. Death has been reported after use of CN in enclosed spaces, but not from use in the open air.

BIOLOGICAL WARFARE: Biological weapons (B.W.) are defined as **microorganisms or their products of metabolism that infect and grow in the target host producing a clinical disease that kills or incapacitates humans or animals.** Such microbes may be natural, wild-type strains or may be

genetically produced. These include biological toxins and substances that interfere with normal behaviour. Bacillus anthracis, smallpox virus, botulinum toxin and ricin are commonly used.

HYDROCYANIC ACID

Hydrocyanic acid (prussic acid or cyanogen) is a solution of HCN in water either 2% or 4%, the latter being called "Scheele's acid". It is a vegetable acid found in nature in many fruits and leaves, such as almond, apricot, apple, cherry, peach, plum, pear, bamboo shoots and in certain oil seeds and beans, where it exists in the form of a glucoside amygdalin, which is harmless, but usually coexists with the enzyme emulsin, which hydrolyses it and liberates hydrocyanic acid. Amygdalin is converted to cyanide in the small intestine by bacteria in the presence of the enzyme emulsin. Hydrocyanic acid is usually obtained from distilling cyanide by an acid. The pure acid is a colourless, transparent, volatile liquid with an odour like bitter almonds and is rapidly decomposed by exposure to light. About 20 to 40% persons cannot smell the gas. Cyanides are white powders. Potassium ferrocyanide and ferricyanide are not poisonous.

Action: Cyanide inhibits the action of cytochrome oxidase, carbonic anhydrase and probably of other enzyme systems. It prevents the formation of ATP and its use as energy source. There is an interference with the intracellular oxidative processes in the tissues and **it kills by creating histotoxic or cytotoxic anoxia, although the blood may contain a normal oxygen content.**

Absorption and Excretion: Cyanide gas is absorbed rapidly from the respiratory system, and the acid and cyanide salts from the stomach. The acid is also absorbed through the skin. **Alkaline cyanides when ingested are converted by hydrochloric acid in the gastric juice into chlorides, and hydrocyanic acid is liberated.** The greater part is converted by an enzyme rhodanese (present in liver and kidney) into thiocyanate. A small amount is eliminated unchanged in the breath. It is mainly excreted in the urine.

Signs and Symptoms: This is most rapid of all poisons. When inhaled as a gas, its action is instantaneous. Massive doses may produce sudden loss of consciousness and prompt death from respiratory arrest. If a large dose is taken, symptoms usually occur at once, but in some cases symptoms appear after about one minute, during which the victim may perform certain voluntary acts, such as corking or throwing away the bottle or walking a little distance. **All symptoms ultimately reflect cellular hypoxia,** and the symptoms shift rapidly, depending on the extent of the cyanide exposure. **CNS:** Headache, vertigo, faintness, perspiration, anxiety, excitement, confusion, drowsiness, prostration, opisthotonus and trismus, hyperthermia, convulsions, epileptiform or tonic, which are sometimes localised but usually generalised, paralysis, stupor, coma, and death. **G.I.T.:** Bitter, acid, burning taste, constriction or numbness of throat, salivation, nausea, rarely vomiting. **R.S.:** Odour of bitter almonds in breath. Initially tachyapnoea and dyspnoea due to cyanide stimulation of chemoreceptors and the respiratory centre. Later rapid slowing of respiratory rate with severe respiratory depression and cyanosis. **C.V.S.:** Initially hypertension with reflex bradycardia, sinus arrhythmia. Later tachycardia with hypotension and cardiovascular collapse. The heart continues to beat for several minutes after stoppage of respiration. **Skin:** Perspiration, bullae. **Eyes:** Glassy and prominent, pupils dilated, unreactive. **Renal:** Acidosis. **Death occurs from respiratory failure.** Survivors of serious acute poisoning may develop delayed neurological sequelae, especially in the form of Parkinsonian symptoms.

Inhalation of vapour produces a sense of constriction about the throat and chest, dizziness, vertigo, insensibility and death from respiratory failure.

Cyanides: In poisoning by cyanides the symptoms may not occur for 10 to 20 minutes because of the delay in the decomposition of the salt by gastric juice, and the liberation and absorption of hydrocyanic acid. Potassium and sodium cyanide have a corrosive effect on the mouth, throat and stomach, and cause epigastric pain and vomiting. Other symptoms are similar to hydrocyanic acid.

Fatal Dose: 50 to 60 mg. of pure acid; 200 to 300 mg. of sodium or potassium cyanide.

Fatal Period: 2 to 10 minutes; sometimes immediate. Potassium or sodium cyanide half hour.

Treatment: No time should be lost in starting treatment. **The principle of the treatment is to reverse the cyanide-cytochrome combination.**

This is achieved by converting haemoglobin to methaemoglobin by giving nitrites. Methaemoglobin has a higher binding affinity for cyanide than the cytochrome oxidase complex, and removes cyanide from the cytochrome oxidase. **Cyanides combine with methaemoglobin and form non-toxic cyanmethaemoglobin, which in the presence of rhodanese and sulphate donors, such as thiosulphate, converts cyanide to thiocyanate which is excreted in the urine.** Cyanide is directly converted to thiocyanate by the complexing of cyanide with thiosulphate under the influence of the enzyme rhodanese. Cyanide is also converted to cyanocobalamin (Vit B_{12}) by complexing with hydroxocobalamin (Vit B_{12} A).

(1) **Break 0.2 ml. ampoule of amyl nitrite in a handkerchief and hold over the patient's nose for 15 to 30 seconds of every minute, until sodium nitrite infusion is started.** (2) **0.3 g. of sodium nitrite in 10 ml. of sterile water is given i.v. slowly, over a period of five minutes.** Sodium nitrite forms methaemoglobin ($Hb–Fe^{2+}$), that competes with cytochrome oxidase for the cyanide ion, thus protecting cytochrome oxidase. Do not remove the needle. (3) Through the same needle **infuse 25 g. of sodium thiosulphate in 50% solution i.v. over a period of ten minutes.** It converts cyanide to non-toxic thiocyanate. Repeat the nitrite-thiosulphate injection after an hour if recovery has not occurred. (4) Both sodium nitrite and sodium thiosulphate can be repeated at half the initial dose at the end of one hour, if symptoms persist or reappear. (5) Other antidotes are: **Hydroxocobalamine** 4 to 5 g. i.v. as infusion. It detoxifies cyanide by giving a hydroxyl group and then binding a cyanyl group from the cyanide, forming non-toxic cyanocobalamin which is excreted in the urine. It may be used with sodium thiosulphate which reacts with cyanocobalamin in the presence of the enzyme rhodanese to produce thiocyanate. (6) **Dicobalt EDTA** acts by chelating cyanide to form a harmless product that is excreted in the urine. 600 mg. is given i.v. slowly. It is followed by 300 mg. if recovery does not occur. Cobalt EDTA and aminophenols are more rapid in action, efficacious, and less toxic than nitrites. (7) 4-dimethyl-aminophenol (4-DMA) 3 mg/kg i.v. (8) Gastric lavage is then performed using activated charcoal, 3% hydrogen peroxide, or 5 to 10% sodium thiosulphate, or 1:5000 potassium permanganate, and 200 ml. left in the stomach. (9) Methylene blue is not effective. (10) Ventilate with 100% oxygen. (11) Methaemoglobin of more than 50% is an indication for exchange transfusion or administration of blood. (12) If death is delayed, a mixture of ferrous and ferric sulphate with potassium carbonate may be given as a chemical antidote to form Prussian blue.

Postmortem Appearances: Eyes may be bright, glistening and prominent with dilated pupils. The jaws are firmly closed and there is froth at the mouth. **The colour of the cheeks and postmortem staining may be cherry-red or brick-red due to the impaired oxygenation of tissues by cyanide** and P.M. formation of cyanmethaemoglobin. The odour of hydrocyanic acid may be noted on opening the body. **In cases of suspected cyanide poisoning, the cranial cavity should be opened first as the odour is usually well marked in the brain tissues. The blood is bright cherry-red in about half the cases**, because oxygen remains in the cells as oxyhaemoglobin, and due to the formation of cyanmethaemoglobin. **All the vessels of the body including the veins contain oxygenated blood.** Bloodstained froth may be found in the trachea and bronchi. There is congestion of viscera (Plate 21-125) and oedema of the lungs. The serous cavities are ecchymosed. The mucosa of the stomach and intestine is often red and congested (Plate 21-123). Hydrogen ajanide can be inhaled from the stomach contents when inspecting viscera. Degenerative changes may occur in the nervous system. **Potassium or sodium cyanide produce slight corrosion of mouth and a diffuse crimson discolouration of gastric mucosa** which gradually changes to rusty-brown due to the formation of alkaline haematin. Other findings are same as that of hydrocyanic acid.

Cyanide may be formed in stored blood samples at room temperature. By contrast in some positive samples, cyanide may decrease on storage.

The Circumstances of Poisoning: (1) Hydrocyanic acid and cyanides are usually used for suicide. **It can be concealed in "suicidal pills", in rings or hollow teeth.** (2) It is rarely used for homicide and as cattle poison. **Cattle poisoning can occur from eating linseed plant due to the natural development of a cyanogenetic glycoside.**

Cyanide is not produced after death by putrefactive changes. Formaldehyde rapidly destroys cyanide.

JUDICIAL EXECUTION: In some countries, hydrocyanic acid gas is used for legal execution. The condemned person is strapped in a metal chair with perforated seat. A bowl with sulphuric acid mixed with distilled water is kept under the chair. Few pellets of sodium cyanide are suspended in a gauze bed just above the bowl. The door is sealed. The executioner seated in an adjacent room operates a lever, which releases cyanide bed into the bowl. This produces large quantity of hydrogen cyanide gas immediately, which rises through the holes in the chair. Unconsciousness takes place very rapidly, although heart continues to beat for 10 to 20 minutes.

CHAPTER

36 Miscellaneous Poisons

ACETYLSALICYLIC ACID (ASPIRIN)

It is a white odourless, crystalline powder, having a slight acid taste. It is used as an antipyretic and analgesic.

SIGNS AND SYMPTOMS: Burning pain in the throat and mouth, anorexia, apathy, lassitude, nausea, vomiting and thirst are the early symptoms. The respiration is at first fast and deep, and later laboured and dyspnoeic. Vertigo, ringing in the ears, impaired vision and deafness are common. Later, metabolic acidosis supervenes. An increased anion gap metabolic acidosis with respiratory alkalosis, ketosis and tinnitus suggests salicylate poisoning. The urine is strongly acid. Hypovolaemia and hypokalaemia may occur. The skin is flushed and moist, pupils dilated, pulse is rapid and irregular. There may be platelet dysfunction and prolonged clotting time. Severe dehydration may occur and in the terminal stages hyperpyrexia of 41 to 42°C may occur. Death occurs from acidosis and uraemia with peripheral failure, due to shock in the earlier stages, or respiratory failure later. Aspirin can cause sudden cardiac arrest in the absence of any toxic symptoms. Fatal cardiac arrhythmias may supervene. Idiosyncrasy is seen in 0.2% persons.

REYE'S SYNDROME: It is sometimes seen in children below 15 years on consumption of aspirin. The main features are acute onset of hepatic failure and encephalopathy.

FATAL DOSE: Aspirin 15 to 20 g; salicylic acid 70 to 80 gm; methyl salicylate 10 to 20 ml.

FATAL PERIOD: Few minutes to several hours.

TREATMENT: (1) Emetics. (2) Stomach wash with 5% sodium bicarbonate. (3) Forced alkaline diuresis. (4) Activated charcoal is useful. (5) Peritoneal and haemodialysis. (6) Exchange transfusion in severe cases. (7) Saline catharsis. (8) Symptomatic.

POSTMORTEM APPEARANCES: Skin rashes may be present. There is generalised congestion of all the organs (Plate 21-122). Petechial haemorrhages are seen in various organs.

Poisoning is almost always suicidal.

INSULIN

It is a white powder with bitter taste.

SYMPTOMS: Weakness, fatigue, vomiting, dizziness, tachycardia, hypotension, cramps, tremors, profuse sweating, tingling, heavy, deep breathing, maniacal behaviour, delirium, shock, coma and death.

TREATMENT: (1) Give 10 to 20 g glucose orally in a solution (or a high carbohydrate food). (2) 50 ml of 50% glucose i.v. Then continue infusion of 10% glucose.

M.L. IMPORTANCE: (1) Impairment of ability to drive a vehicle. (2) Homicide is very rare. (3) Hypoglycaemia (non-insane automatism).

NITRITES

Inorganic nitrite salts and organic nitrites and nitrates are available in many houses as medicines, e.g. amyl nitrite, nitroglycerine, sodium nitrate and spirits of niter. Sodium nitrate is used as a mordant by weavers.

Action: Sodium nitrate causes relaxation of smooth muscle, especially of small blood vessels and in toxic doses converts haemoglobin to methaemoglobin.

Fatal Dose: Sodium nitrate one to two g; nitroglycerin 200 mg.

Fatal Period: Few hours to few days.

Symptoms: Low blood pressure, throbbing headache, vertigo, palpitations, visual disturbances; skin is flushed and perspiring, later cold and cyanotic; nausea, vomiting, colic, bloody diarrhoea; syncope, methaemoglobinaemia, cyanosis and anoxia; hyperpnoea and later dyspnoea; slow pulse; raised intracranial pressure and intraocular tension; paralysis, coma followed by clonic convulsions; death due to circulatory collapse.

Treatment: (1) Stomach wash with potassium permanganate. (2) Methylene blue one percent solution, one to two mg/kg i.v., or fifty mg/kg orally converts methaemoglobin to hemoglobin. (3) Transfusion with whole blood or plasma expanders. (4) Oxygen and artificial respiration.

Autopsy: Signs of asphyxia are seen. Blood and postmortem lividity are chocolate coloured. Sodium nitrate deteriorates rapidly after death.

Poisoning: Poisoning is usually accidental. Poisoning occurs in infants fed with well water contaminated with nitrates.

METHAEMOGLOBINAEMIA: Methaemoglobin is hemoglobin in which the normally ferrous iron of the haeme moiety has been oxidised to the ferric state, making it incapable of oxygen transport. The shape of oxygen-haemoglobin dissociation curve is altered, aggravating cellular hypoxia (Table 36-1).

Table 36-1. Symptoms associated with methemoglobin concentration

Met Hb Conc%	Symptoms
0–3	Normal level.
3–15	No symptoms; slate grey skin.
16–20	Cyanosis, chocolate-brown blood.
21–50	Dyspepsia, exercise intolerance, headache, fatigue, dizziness, syncope, weakness.
51–70	Tachypnoea, metabolic acidosis dysrhythmias, convulsions, CNS depression, coma.
>70	Severe hypoxic symptoms.

CAUSES: (1) Hereditary: Haemoglobin M, nicotinamide adenine dinucleotide methaemoglobin reductase deficiency. (2) Acquired: (a) Direct with nitrites, food high in nitrates, nitroglycerine, nitrous gases, chloroquine, primaquine, silver nitrate, well water (nitrates). (b) Indirect oxidants: aniline dye derivatives, benzocaine, sulphonamides, chlorobenzene, dapsone, naphthalene, nitrophenol, nitroprusside, phenacetin, pyridium, trinitrotoluene, bromates, chlorates, pyrogallol.

TREATMENT: (1) High-flow oxygen. (2) Activated charcoal. (3) Methylene blue, i.v., 1 to 2 mg/kg as one % solution slowly over 5 minutes. If cyanosis does not disappear within one hour, a second dose should be given.

ANTIHISTAMINICS

The commonly used preparations are: antazoline, (antistine), diphenhydramine (benadryl), alopyramine hydrochloride (synopen), mepyramine maleate (anthisan), and promethazine hydrochloride (phenergan). Other preparations are: tripelennamine, chlorpheniramine, cimetidine, ranitidine, nizatidine and famotidine.

Fatal Period: 1 g.

Fatal Dose: Uncertain.

SYMPTOMS: In adults, CNS depression is the usual dominant reaction characterised by drowsiness, lethargy, fatigue, hypnosis and coma. There is vertigo, ataxia, tinnitus, dilated pupils and blurred vision. The initial sedation is often followed by CNS hyperexcitability; sometimes the excitement is the first evidence of poisoning. This causes tremors, anxiety, insomnia, excitement, delirium and convulsions. Anticholinergic features (mydriasis, hyperthermia and flushing) are seen. Gastrointestinal symptoms are: dry mouth, anorexia, nausea, vomiting, abdominal pain, constipation or diarrhoea. There may be loss of balance, hallucinations, tachycardia, retention of urine and skin rashes. Finally, there is severe central nervous depression, and death results from respiratory failure or cardiovascular collapse.

Poisoning is usually accidental and sometimes suicidal. One gram is fatal.

TREATMENT: (1) Stomach wash, (2) Activated charcoal. (3) Diazepam. (3) Physostigmine 0.5 to 2 mg. i.v. every hour, until reversal of symptoms occur. However, it can produce serious adverse effects. (4) Symptomatic.

AUTOPSY: Signs of asphyxia are found.

CAFFEINE

Caffeine stimulates gastric acid, pepsin and secretions from small intestine. Large doses can stimulate directly the myocardium to produce tachycardia, arrhythmias and extrasystoles. It increases cardiac output and stroke volume. It decreases fatigue. Adrenaline and noradrenaline secretion is increased. It can increase the basal metabolic rate by about 10%. It also increases oxygen consumption. It acts as diuretic. Significant amounts of caffeine are present in tea, cola beverages and chocolates.

FATAL DOSE: 1 to 2 g.

Doses of 50 to 200 mg. result in increased alertness, decreased drowsiness and lessened fatigue. Doses of 200 to 500 mg. may produce headache, tremors, nervousness, irritability and slight increase of blood pressure. At level of one gram (8 to 10 cups of coffee) per day, a combination of physiological and behavioural symptoms like anxiety-like presentation, insomnia, headache and depressive presentation can appear. With 10 g. of caffeine, grand mal seizures, and cardiorespiratory arrest may occur.

Withdrawal symptoms are: headache, yawning, nausea, drowsiness, lethargy, rhinorrhoea, irritability, nervousness and depression.

FORMALDEHYDE

It is a colourless gas having a strong, pungent, irritating odour. 40% solution in water is known as formalin. Commercial formalin contains 37% formaldehyde and 10 to 15% methanol. It is readily absorbed in upper respiratory tract. It is metabolised to formic acid in liver and blood.

SYMPTOMS: Inhalation of vapour causes burning of eyes, lachrymation, coughing, constriction in chest

and palpitation. Ingestion produces symptoms similar to strong acid. It can cause contact dermatitis. Chronic exposure can result in allergic contact dermatitis, chronic obstructive pulmonary disease and optic neuritis. It is used as disinfectant, for preservation of museum specimens, in plastic, in dyeing, hardening of celluloid and as a reducing agent. It gives off vapour at room temperature.

FATAL DOSE: 30 to 60 ml.

FATAL PERIOD: 1–2 days.

TREATMENT: (1) Wash the stomach with 0.1% solution of ammonia, as it reacts with formaldehyde to form harmless methenamine. (2) Symptomatic.

POSTMORTEM APPEARANCES: Smell is noted on opening the body. Mucosa of the stomach may be red, inflamed and eroded with extravasation of blood, or it may be hard and tough like leather. The intestines and lungs are congested. The liver may show fatty degeneration and the kidneys may be inflamed.

PARACETAMOL (ACETAMINOPHEN)

It is absorbed rapidly from the gastrointestinal tract, and metabolised quickly in the liver. It is a potent hepatic toxin. A small part is converted by a liver enzyme into N-acetyl-p-benzoquinoneimine. Glutathione and other sulfydryl compounds detoxify this substance, but in overdose NABP accumulates and causes severe centrilobular liver necrosis. The simultaneous presence of phenobarbitone or phenytoin in epileptics or the presence of chronic alcoholism greatly worsens the situation. Large doses act on brain stem and cause rapid death. Most deaths are delayed for several days, when liver failure occurs. Toxic doses cause depletion of glutathione, which results in hepatic necrosis.

Fatal Dose: 20 to 25 g.

Fatal Period: 3 to 4 days.

Symptoms: Within a few hours, patient experiences anorexia, nausea, vomiting, abdominal pain, diaphoresis, hypotension, tachycardia and dyspnoea. After 1 to 2 days, the discomfort disappears. After 2 to 4 days there is vomiting, jaundice, hepatic pain, bleeding, hypoglycaemia, confusion, coma, metabolic acidosis and coarse flapping tremor of hands (asterixis). There may be cardiac arrhythmias, haemorrhagic pancreatitis, disseminated intravascular coagulation, etc. Death usually occurs in 3 to 4 days. Renal failure due to renal papillary necrosis may occur in 24 to 72 hours even in the absence of hepatotoxicity. Death from hepatic failure occurs 4 to 18 days postingestion. Paracetamol has replaced barbiturates and aspirin in Britain as favourite method of suicide.

Treatment: (1) Gastric lavage. (2) Activated charcoal. (3) **N-acetylcysteine (NAC) is a specific antidote** and has maximum efficacy if used within 8 hours. It is given in a dose of 140 mg/kg body weight. Then 70 mg/kg is given every 4 hours until a total of 18 doses over 72 hours period orally. It can also be given i.v. 150 mg/kg in 200 ml of 5% dextrose over 15 minutes, followed by 50 mg/kg in 500 ml of 5% dextrose over 4 hours, and 100 mg/kg in one litre over 16 hours. (4) Methionine is less effective than NAC. It acts by increasing glutathione synthesis. Initial dose is 2.5 g. orally, repeated every four hours up to a total of 10 g. (5) Correct acidaemia. (6) Haemodialysis. (7) Symptomatic.

Postmortem Appearances: They include acute centrilobular hepatic necrosis, acute tubular necrosis in the kidney, myocardial necrosis, and cerebral oedema.

WATER INTOXICATION

Deaths from water intoxication are very rare. Deaths occur due to ingestion of large quantities of water or the administration of large quantities of i.v. fluids, devoid of electrolytes. The victims are usually psychotic. Death occurs due to cardiac arrhythmia produced by electrolyte imbalance.

Postmortem diagnosis should be based on history and low levels of sodium and chlorides in vitreous fluid. Potassium levels are normal or high, as it is rapidly released from the cells of the body after death, even in the vitreous.

CHAPTER

37

Food Poisoning

FOOD POISONING: The term food poisoning in its wider sense includes all illnesses which result from ingestion of foods containing non-bacterial products. But the term is usually restricted to **acute gastroenteritis due to the bacterial infection of food or drink. WHO** defines food poisoning as **"diseases usually either infectious or toxic in nature, caused by agents that enter the body through the ingestion of food".**

BACTERIAL FOOD POISONING:

(1) SALMONELLA FOOD POISONING: Man gets the infection from farm animals and poultry through contaminated meat, milk and milk products, sausages, custards, egg products. Rats and mice are another source. The causative organisms, e.g., S. enteritidis of Gaertner, S. typhimurium, S. choleraesuis, and less commonly paratyphoid bacilli, on ingestion, multiply in the intestine and give rise to acute enteritis and colitis. Mortality is about 1 per cent. Incubation period is 12 to 24 hours.

(2) STAPHYLOCOCCAL FOOD POISONING: Enterotoxins of certain strains of coagulase-positive Staphylococcus aureus is the causative agent. The foods involved are salads, custards, milk and milk products which get contaminated by staphylococci. Incubation period is 1 to 6 hours. Food poisoning results from ingestion of toxins, e.g., enterotoxin, preformed in the food in which bacteria have grown (intradietetic toxins). Since the toxin is heat-resistant, it can remain in food after the organisms have died. The toxins act directly on the intestine and CNS. The illness becomes manifest by the sudden onset of vomiting, abdominal cramps and diarrhoea. The patient recovers as soon as the enterotoxins have been neutralised and metabolised usually within 24 hours of poisoning. ~~Death~~ is uncommon.

(3) CI. PERFRINGENS FOOD POISONING: The organism has been found in faeces of humans and animals, and in soil, water and air. The majority of outbreaks have been associated with the ingestion of meat, meat dishes and poultry. The food has been prepared and cooked 24 hours or more before consumption, and allowed to cool slowly at room temperature and then heated immediately prior to serving. The spores are able to survive cooking. The most common symptoms are diarrhoea, abdominal cramps and little or no fever, occurring 8 to 24 hours after consumption of the food. Nausea and vomiting are rare. Illness is usually of short duration, of 1 day or less. Recovery is rapid and no ~~death~~s have been reported.

(4) B. CEREUS FOOD POISONING: Bacillus cereus is ubiquitous in soil, and in raw, dried and processed foods. The spores can survive cooking and germinate and multiply rapidly when the food is held at favourable temperatures. B. cereus produces at least 2 distinct enterotoxins, causing 2 distinct forms of food poisoning. (1) The emetic form characterised by predominantly upper gastrointestinal tract symptoms. (2) The diarrhoeal form characterised by predominantly lower intestinal tract symptoms like CI perfringens food poisoning. Recovery within 24 hours is usual. The toxins are preformed and stable.

Food poisoning is common in summer, because the temperature favours multiplication of microorganisms. It may occur as isolated cases or small outbreaks. The main diseases spread by infected food are: (1) The enteric group. (2) Cholera. (3) Bacillary dysentery. (4) Staphylococcal and other bacterial infections. (5) Amoebic dysentery and other protozoal infections. (6) Acute infective hepatitis. (7) Brucellosis. (8) Various worm infestations. (9) Schistosomiasis. (10) Traveller's diarrhoea (due to pathogenic E.coli).

BOTULISM: There are no or very slight symptoms of gastroenteritis although poisoning results from absorption of a specific toxin from the alimentary canal. Cl. botulinum does not grow in body but produces a potent neurotoxin. It is normally present in the soil, dust and the intestinal tract of animals and enters food as spores, especially fruits and vegetables. The toxin is destroyed by heat at 80°C. Botulism is an intoxication, not an infection. The causative organism C. botulinum multiplies in the food, e.g., tinned meat, fish, fruits, etc., before it is consumed and produces a powerful exotoxin under suitable anaerobic conditions. The fatal dose for an adult is 0.01 mg. or even less. The toxin paralyses the nerve endings, by blocking the nerve impulses at the myoneural junctions. It blocks the action of acetylcholine. Its action is selective being confined to the cholinergic fibres of the autonomic nervous system.

Action: Its action is selective being confined to the cholinergic fibres of autonomic nervous system. It affects peripheral cholinergic nerve endings including neuromuscular junction, postganglionic parasympathetic nerve endings and peripheral ganglia without affecting CNS.

Signs and Symptoms: The incubation period varies from 12 to 36 hours but may be prolonged to 72 hours. Symptoms are mainly due to the action of the toxin on the central nervous system. The initial G.I. symptoms include nausea, vomiting, abdominal distension and pain. Later symptoms are: dry or sore mouth or throat, difficulty with visual accommodation, dysphonia, diplopia, ptosis, dysarthria, blurring of vision, muscle weakness, descending bilaterally symmetrical motor paralysis, dysphagia, constipation, respiratory insufficiency and urinary retention. The patient is conscious until death, which is preceded by coma or delirium. The temperature is usually subnormal. Death may occur due to respiratory or cardiac failure, within 24 hours from the onset of symptoms but may be delayed for a week. Mortality varies from 25 to 100%. At autopsy the kidneys, liver and meninges are congested.

Treatment: (1) Gastric lavage or emesis. (2) Activated charcoal. (3) Purgative. (4) Whole bowel irrigation. (5) Botulinum antitoxin (types A, B and E) one vial (50,000 to 1,00,000 units) by slow i.v. in normal saline and one vial i.m. and repeated at 2 to 4 hour interval i.v. (6) Botulism immune globulin (BIG) is a pentavalent types A, B, C, D and E 50 ml is given. (7) Guanidine 15 to 40 mg/kg/day orally reverses the neuromuscular block of botulism.

Viruses: Viral infections usually have 1 to 3 days incubation period and are self-limited and similar to the bacterial type. Norovirus and rotovirus commonly cause food poisoning.

FOOD ALLERGY: It is not food poisoning, for in this the abnormality is not in the food but in the allergic person. Some persons are hypersensitive to certain types of protein, e.g., meat, fish, eggs, milk, etc. which are ordinarily quite harmless and suffer from gastroenteritis, local urticarial rashes or asthmatic attack.

PTOMAINES: These are alkaloidal bodies which are formed as the result of bacterial decomposition of protein. When they are formed in the dead tissues, they are known as cadaveric alkaloids. Alkaloids secreted by living cells during metabolism are called leucomaines, which are slightly toxic when injected into an animal, but have no action when ingested. They are not bacterial poisons and are not derived from bacteria. They are found only when the food becomes too disagreeable to be eaten. Most of the ptomaines are non-poisonous except neurine and mydaleine, which are produced in traces 5 to 7 days after death. The symptoms resemble that of atropine. Ptomaines are not the causative agents of food poisoning.

Mycotoxicosis: The spores of moulds grow on the food of man and animals and release highly toxic mycotoxins. They can remain in meat and be passed into milk or eggs. Mycotoxins are heat stable and survive cooking. The consumption of contaminated food causes poisoning in man. Aspergillus flavus can grow on any food. They produce aflatoxins. It can produce carcinoma of liver and acute encephalitis. The fungus penicillium islandicum growing on yellowed rice produces islandotoxin which is a potent hepatotoxin.

POISONOUS FOODS

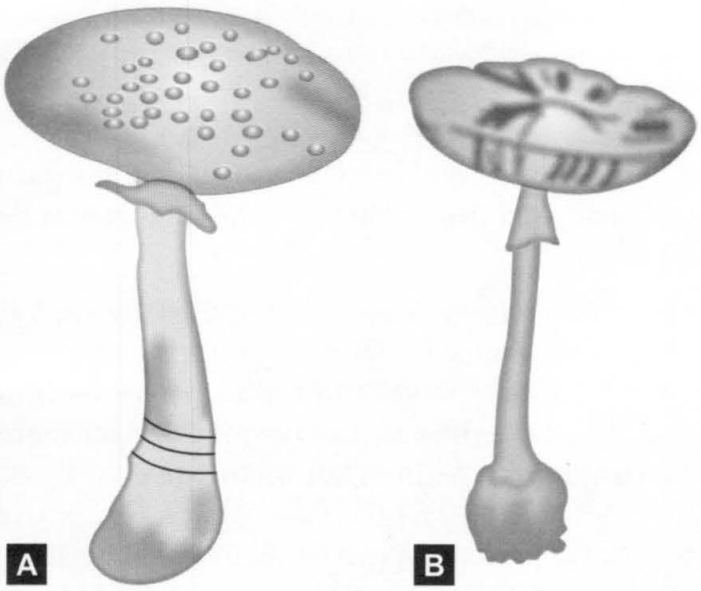
Poisonous foods are those which contain poison derived from plants, animals and inorganic chemicals.

LATHYRUS SATIVUS (KESARI DHAL): It is a staple food for the low income groups in some areas of Central India. **Consumption of L. sativus seeds in quantities exceeding 30% of the total diet for more than 6 months have been known to cause paralysis.** Men are more susceptible than women. The active neurotoxic principle is B(N) oxalyl aminoalanine (BOAA), which is present as a free amino acid in the seed cotyledons to the extent of about 1%.

Onset is of three types: (1) acute, (2) subacute, and (3) insidious. The continued use of L. sativus **produces neurolathyrism, which is characterised by progressive spastic paraplegia** with preservation of sphincters, sensation and mental activity. It progresses irreversibly into 4 stages of physical disability.

MUSHROOMS: Some species are non-poisonous and are used as food. Amanita muscaria (Fig. 37-1A) and Amanita phalloides (Fig. 37-1B) are the common varieties of poisonous fungi. Poisonous mushrooms usually have a bitter, astringent, acid or salt taste, and on section and exposure change colour; a brown, green or blue colour developing on the cut surface. Amanita muscaria contains an alkaloid muscarine, the action of which resembles stimulation of parasympathetic post-ganglionic nerves. Amanita phalloides is also called the deadly agaric or death cap. The fungus is a powerful poison and **contains phalloidin, phallon, and B amanatin, which are cyclopeptides and virotoxins.** These polypeptides are heat stable and insoluble in water. They are powerful inhibitors of cellular protein synthesis. Muscarine stimulates post-ganglionic cholinergic fibres.

Symptoms: In some cases irritant symptoms may be present, and in others neurotic or a combination of both. The irritant symptoms are delayed for 6 to 12 hours. There is constriction of the throat, burning pain in the stomach, nausea, vomiting and diarrhoea followed by cyanosis, slow pulse, laboured respirations, convulsions, sweating,



Figs (37-1A and B). Mushrooms: (A) Amanita muscaria; (B) Amanita phalloides.

collapse and death. The neurotic symptoms are giddiness, headache, delirium, diplopia, constriction of pupils, cramps, twitching of the limbs, salivation, bradycardia and coma. Hepatic and sometimes renal toxicity occurs between 3 days and 6 days.

Fatal Dose: 2 to 3 mushrooms.

Fatal Period: Usually 24 hours.

Treatment: (1) Stomach wash with potassium permanganate. (2) Activated charcoal. (3) Forced diuresis. (4) Benzyl penicillin 3 lakhs to one million units daily. (5) Atropine sulphate. (6) Anti-phalloidien serum. (7) Haemodialysis.

Autopsy: Inflammation of the mucous membrane of the alimentary canal, fatty degeneration of the liver, kidneys and heart may be found. In case of neurotic symptoms, congestion of the brain and petechial haemorrhages in serous membranes are seen.

ARGEMONE MEXICANA: (*ujarkanta; kutila; sialkanta*): It grows wild all over India in the cold season **(Plate 24-149)**. The seeds are dark-brown in colour, globular, smaller than mustard seeds and covered with minute, regularly arranged projections, and depressions. **The plant contains 2 alkaloids berberine and protopine. The oil contains 2 alkaloids, sanguinarine and dihydrosanguinarine.** All parts of the plant are poisonous. **The oil causes epidemic dropsy**. The oil from the seeds is sometimes used as an adulterant of mustard oil, or other edible oil. It causes abnormal permeability of blood vessels.

Symptoms: Symptoms appear slowly with loss of appetite, diarrhoea, marked oedema of the legs, sometimes generalised anasarca. In severe cases, myocardial damage and dilatation of the heart is seen. Blood pressure is low and pulse feeble and rapid. Liver may be enlarged and tender. Patient becomes breathless. Tingling and hyperaesthesia of skin and tenderness of the calf muscles may be seen. The jerks are feeble or absent. Dimness of vision is seen in about 10% of cases due to increased intraocular pressure of glaucoma. Bluish mottling of the skin is seen due to dilation of the peripheral vessels. Death occurs from severe damage to the heart.

Stigmata maides (maize) causes pellagra when eaten, due to lack of nicotinic acid.

Cotton seeds have toxin gossypol (cake) which makes lysine unavailable to the body.

FISH AND MARINE ANIMALS: Ingestion of fish causes two types of poisoning. The first is due to bacterial growth in partially decomposed fish. The other is a primary toxicity caused by the presence in certain fishes of a neurotoxin. 99% of cases of fish poisoning are **icthyosarcotoxic or icthyotoxism** (involving toxins from muscles, viscera, skin, gonads and mucous surfaces). Rarely, toxicity involves the fish blood or skeleton.

Ciguatera poisoning is the most common accounting for 50%.

Venomous Fish: Some fish such as cat fish, muraena (eel), dragon fish, lion fish, etc. have spine, which may cause poisonous wounds. Several families of bony fish have venom glands enveloping sharp spines, which may be dorsal, ventral, lateral, behind the head or on the tail.

Appendix

THE WORKMEN'S COMPENSATION ACT, 1923: This Act provides for the payment of compensation to workmen for injuries sustained by them by accident, arising out of and in the course of employment. If a workman is killed, his dependants will be entitled to compensation for his death. Under the Act, if a workman contracts any disease specified therein as an occupational disease peculiar to that employment (anthrax, primary cancer of the skin, pathological manifestations due to X-rays, radium, etc., poisoning by lead, phosphorus, mercury, arsenic), it is deemed to be an injury by accident for purpose of compensation. The amount of compensation depends upon whether the injury has caused death, permanent total disablement, or permanent partial disablement.

In all industrial diseases and injuries, medical evidence will be necessary. As such, it is important for a doctor to keep complete records of any injury sustained by an employee during the course of employment. It should be determined whether or not there is any causal relationship between an accident or injury and a death, and of acceleration or aggravation of a pre-existing natural disease process. He should diagnose and evaluate the presence and extent of occupational disease.

CONSUMER PROTECTION ACT, (CPA/COPRA) 1986

This Act provides for better protection of the interests of the consumers. The Act covers all private, corporate and public sector enterprises.

CONSUMER DISPUTES REDRESSAL AGENCIES: (1) District Forum entertains claims up to 20 lakhs. (2) State Commission entertains claims between 20 lakhs to one crore. (3) National Commission entertains claims of over rupees one crore.

They provide speedy and simple solution to consumer's disputes. These quasi-judicial bodies observe the principles of natural justice, and are empowered to give reliefs of a specific nature, and award wherever appropriate compensation to consumers. It also prescribes penalties for non-compliance of the orders.

MANNER IN WHICH COMPLAINT SHALL BE MADE: A complaint, in relation to any goods sold or delivered or any service provided or agreed to be provided may be filed with a District Forum by (a) consumer, (b) recognised consumer association, (c) one, or more consumers, (d) Central or State Government.

PROCEDURE: Every complaint is heard as early as possible. If the complaint relates to any services, the District Forum shall refer a copy of such complaint to the opposite party within 21 days of the admission date, directing him to give his version of the case within a period of 30 days. It is not necessary for the parties to be represented by lawyers. The dispute is settled on the basis of evidence brought to its notice by the complainant and the opposite party. The District Forum has the same powers as are vested in a Civil Court (First Class Magistrate), under the Code of Civil Procedure, 1908. Ordinarily, no adjournment is granted unless sufficient cause is shown. If the District Forum is satisfied that the goods complained against suffer from any of the defects specified in the complaint or that any of the allegations contained in the complaint about the services are proved, it will issue an order to the opposite party directing him to remove the defect or replace the goods, or to provide adequate costs to parties.

APPEALS: The aggrieved person can prefer an appeal to the State Commission within a period of thirty days from the date of the order. Further appeals can be made to National Commission and finally to the Supreme Court, within a period of 30 days from the date of the order.

CPM AND MEDICAL SERVICES: The doctors and hospitals that render service as medical practitioners are liable for any act of 'medical negligence' and they can be sued for compensation under the Consumer Protection Act. Service rendered to a patient by a medical practitioner by way of consultation, diagnosis and treatment, both medical and surgical, at a Government hospital, health centre or dispensary, or at a non-government hospital or nursing home where charges are required to be paid by all persons or persons who are in a position to pay, and the persons who cannot afford to pay are rendered free of charge, fall within the purview of this Act. Service rendered at a government hospital, health centre or dispensary, or at a non-government hospital or nursing home by a doctor attached or employed in a hospital or nursing home where no charge, whatsoever, is made from any person availing the services and all patients (rich and

poor) are given free service is outside the purview of the Act. The payment of token amount for registration purpose only at the hospital or nursing home would not alter the position.

Doctors are liable to pay compensation to the patient, if their professional negligence results in injury or death of the patient. As there is no scope for testimony by medical experts, and the District Forum or State Commission comes to its own conclusions, there is every likelihood of the justice being miscarried. A large number of frivolous cases are also likely to be filed against the doctors.

HUMAN EXPERIMENTATION

World Medical Association adopted a Code of Ethics on human experimentation in 1964. Human experimentation may be: (1) Therapeutic: It is concerned with improving the condition of a patient under treatment. (2) Research: It is concerned with using a human being as a means of expanding scientific knowledge for the benefit of humanity. (3) Innovative: They are therapeutic procedures that have not yet earned a place in medical practice.

Biomedical research involving human subjects must conform to the moral and scientific principles that justify medical research, which should be based on scientifically established facts and animal and laboratory experiments. The doctor must be free to use a new therapeutic measure which is likely to save patient's life, reestablish health or alleviate suffering and may be continued if justified by its therapeutic value for the patient. In a purely scientific clinical research, the involved risks should be explained and an informed written consent taken from him, with a proviso of his right to withdraw from the investigation whenever he likes.

It would be unethical to do something which is not strictly related to the cure of the patient's illness. Also, there should not be great risk in the proposed experimentation, and it should be stopped immediately if any ill-effect is noted, which should be treated.

THE PRENATAL DIAGNOSTIC TECHNIQUES (P.D.T.) ACT, 1994.

The objects of the Act are: (1) Prohibition of the misuse of prenatal diagnostic techniques for determination of sex of foetus, leading to female foeticide. (2) Prohibition of advertisement of P.D.T. for determination of sex. (3) Permission and regulation of the use of PDT for the purpose of detection of specific genetic abnormalities or disorders, only on certain conditions by the registered institutions.

The tests should not be carried out unless any one or more of the following conditions are fulfilled, namely: (1) pregnant woman is above 35 years, (2) history of two or more spontaneous loss of pregnancy, (3) history of exposure to teratogenic drugs, radiation, infections, or hazardous chemicals, (4) family history of mental retardation or physical deformities or any other genetic disease. The tests can be carried out only for the detection of: (1) chromosomal abnormalities, (2) genetic or metabolic disorders, (3) haemoglobinopathies, (4) sex-linked genetic diseases, (5) congenital anomalies. State Appropriate Authority advises the Government on policy matters. A State Vigilance Committee and Local Vigilance Committees are constituted.

BIOMEDICAL WASTE (MANAGEMENT AND HANDLING) RULES, 1998 AND 2000

The wastes produced may be of the following categories:

(1) **General waste.**
(2) **Chemical waste.**
(3) **Clinical waste.**
(4) **Radio-active waste.**
(5) **Pressurised containers.**

The amount of waste generated per bed per day varies between 1 to 5 kg and the wastes may be:

(1) **Non-infectious—80% to 85%**
(2) **Infectious—10%**
(3) **Hazardous—5%.**

Every State including Union Territory will establish Prescribed Authority with such members as is specified in the Rules, for authorisation and implementation of the Rule.

As per the rules, Government will also constitute an Advisory Committee comprising of experts in the field of medical and health, animal husbandry and veterinary science, environmental management, municipal administration and other related departments or non-government organisations, along with representative from State Pollution Control Board/ State Pollution Control Committee. This committee will suggest the government and the prescribed authority for implementations of the Rules.

Rules for management and handling of bio-medical wastes:

(1) Biomedical wastes cannot be mixed with any other wastes.
(2) The waste should be segregated in separate container with proper labelling at the point of generation of such wastes and colour code of such containers are as follows:
   (a) Black — For food, paper, dry waste.
   (b) Blue — For crushed syringes, needles.
   (c) Red — For I.V. sets, urobags, catheters.
   (d) Yellow — For dressing materials, body parts, placenta, plaster castes, etc.
(3) No biomedical waste not to be kept stored beyond 48 hours without the permission of the prescribed authority.

(4) Transportation of such wastes can be done in such vehicles authorised for the purpose by competent Authority specified by the Government.
(5) Wastes should be disposed of by:
  (a) Incineration.
  (b) Autoclave.
  (c) Micro-oven system.
  (d) Common waste treatment facility.
  Prescribed authority sets up the standards for autoclaving, incineration, deep burial, micro-ovening for liquid waste and for disposal of treatment of bio-medical waste.
(6) Proper recording of such disposal is compulsory.

The prescribed authority/environment pollution control board is conferred with the power of inspection and verification of such acts.

TRANSPLANTATION OF HUMAN ORGANS ACT, 1994

Central and State Governments are empowered to appoint Appropriate Authority who will grant registration, cancellation, renewal or supervision of the hospitals dealing with organ transplantation and also specify the criterias/conditions required to be fulfilled for registration of such organisations.

Authorisation committee will impose restriction of removal and transplantation of human organs. Human organs cannot be removed for any purpose other than therapeutic purpose.

Purpose of the Act:

(1) To legalize and regulate removal of human organs for transplantation and storage, both from living and dying persons (living cadaver) where brain stem death has occurred.
(2) To declare brain stem death a board of medical experts is permitted to certify. The board consists of
  (a) Medical officer-in-charge of the hospital.
  (b) An independent medical specialist.
  (c) A neurologist/neurosurgeon.
  (d) The treating physician of the patient.
(3) For testing the acceptability of foreign organs/tissue by the recipient, the recommended guidelines are to be followed.
(4) The hospital where transplantation will be performed should have compulsory registration fulfilling the conditions laid down in guidelines.
(5) The laboratories where the tests will be done, should have proper facilities and certified by Appropriate Authority.
(6) After removal of the organ, it is to be stored following current and accepted scientific method to retain vitality.

Penal Provision of the Act:

Punishment includes erasure of the name of the physician for 2 years from Provincial Medical Council for the first offence and permanent erasure of the name of subsequent offences, or imprisonment for 2 to 5 years with fine of ₹ 10,000/- depending upon the nature, character and degree of offence.

For commercial trading of human organs, the offender is liable to be punished for an imprisonment up to 7 years and fine up to ₹ 20,000/-. The cases under this Act are tried by First Class Judicial/ Metropolitan Magistrates.

WOUND CERTIFICATE

Instructions: (1) Obtain written consent for examination and investigations. (2) Examine without delay at any time of day or night. (3) If treated as outpatient, it should be noted in the report. (4) Admit if injuries are serious. (5) If death is imminent, arrange for dying declaration. (6) Note at least two identification marks, preferably on exposed areas of the body. (7) Carry out immunisation when necessary. (8) All forms filled for laboratory investigations should be labelled by the words MLC. (9) Inform police before discharging against advice. (10) In case of discharge or death in hospital, inform the police without delay and do not issue death certificate. (11) If a person is brought dead to the hospital, do not examine wounds, inform police and do not hand over the body to the relatives. Fill death certificate form or note on the out-patient ticket without giving cause of death. (12) If the investigating officer gives requisition for any clarifications regarding any points in the report, the answers should be given in writing (S. 197, I.P.C. and S. 161, Cr.P.C.). (13) Oral statements made to the police and recorded by the police, should not be signed (S. 162, Cr.P.C.). (14) If the investigating officer requires any documents regarding the injured person, either original or photocopy, it should be given and a receipt obtained. (15) The doctor can be summoned to police station for recording a statement if the investigation demands (S. 160, Cr.P.C.).

Wounds or injuries found on the person of a male/ female calling himself/herself ..... aged ..... years, an inhabitant of ..... who was sent with (letter/memo No.) ..... dated ..... from P.S. ..... and accompanied by (name, number of P.C. of P.S.) ..... for report as to certain injuries ..... said to have been caused on ..... and to be due to .....
Identification marks: (1) ..... (2) .....
Consent of the individual for examination .....
Signature or thumb impression of the person
Identified by constable No. ..... Name .....
..... P.S. .....

The injured person was first seen by the undersigned at ..... A.M./P.M. on (date) and the examination was commenced at ..... A.M./P.M. on (date) when the following injuries were found:

1	2	3	4	5	6	7
Nature of injury, i.e. whether an abrasion, cut, bruise, burn, stab, fracture or dislocation, etc.	Size of each injury in cms. i.e., length, breadth and depth	On what part of the body inflicted	Whether simple or grievous	By what kind of weapon inflicted	Whether the weapon was dangerous or not	Remarks, (age of wound, in-patient or outpatient, etc.)

Opinion: I am of opinion that (injuries are simple, grievous or likely to be fatal) .....

Station: Signature:

Date: Name:

Designation:

AGE ESTIMATION

Instructions: (1) Obtain written consent for examination and for taking X-rays. (2) For confirmation of age as determined by dental examination, take X-rays of: (a) Between 6 and 12 years: elbow and wrist joints. (b) Between 13 and 16 years: elbow and pelvis. (c) 16 to 17 years: elbow and ankle. (d) 17 to 18 : hip. (e) 18 to 19 : knee and shoulder. (f) 18 to 20 : iliac crest. (g) 21 : ischial tuberosity and inner end of clavicle. (3) All forms filled for X-rays should be labelled by the words MLC. (4) Note at least two identification marks, preferably on exposed areas of the person. (a) In the case of mole, note colour, size, shape, hairy or not raised or not, and exact anatomical location. (b) In case of scar, note colour, length, width, shape, fixed or free, smoothness or irregularity of surface, presence or absence of glistening and tenderness, direction and exact location. (c) In case of tattoo mark, note colour, design, size and situation and make a sketch.

Requisition from ..... of police of P.S. ..... vide his letter No. ..... dated ..... and accompanied by (name and number of P.C.) of P.S. ..... for determination of age.

(1) Name of the individual:
(2) Sex:
(3) Parent's or guardian's name:
(4) Address:
(5) Occupation:
(6) Caste:
(7) Married or single:
(8) Age as alleged by:
  (a) Individual to be examined:
  (b) Person or police accompanying:
(9) Person accompanying or brought by:
(10) Time and place of examination:
(11) Consent of the individual for examination:
(12) Signature of the individual consenting or his/her left thumb impression:
(13) In the case of minors, consent of the guardian and his/her signature or left thumb impression:
(14) Name of female attendant/nurse present at the time of examination:
(15) Date and time of examination:
(16) Marks of identification: (1) (2)

Physical Examination

(1) Height:
(2) Weight:
(3) Chest girth at the level of the nipples:
(4) Abdominal girth at the level of the navel:
(5) General build and appearance:
(6) Voice:
(7) Teeth:

R (S) 8 7 6 5 4 3 2 1 (S) 1 2 3 4 5 6 7 8

L (S) 8 7 6 5 4 3 2 1 (S) 1 2 3 4 5 6 7 8

T: Temporary P: Permanent

(S): Space after teeth

(8) Hair: Scalp:
Beard: Moustache:
Axillary: Pubic: Body:
(9) Mammae: Development of breasts: milk in
(10) Generative organs:
Development of genitals
(11) Onset of puberty:
(a) Date of menarche:
(b) Regularity of menses:
(12) Date of sending case for radiological examination and P.C.No.:
(13) Number and region of X-rays taken:
(14) Date of receiving the report from the radiologist:

(15) Radiological findings:

Opinion of age:

Station: Signature:
Date: Name:
Designation:

AGE CERTIFICATE

Name of individual: ..... Sex .....
Cr. No. ..... of P.S. ..... dated .....
accompanied by P.C.No. .....

From physical, dental, and radiological examination of ..... bearing the identification marks: (1) ..... (2) .....

Opinion:

I am of opinion that the individual is aged about ..... years.

Station: Signature:
Date: Name:
Rank:

PROFORMA FOR EXAMINATION OF PERSONS FOR DRUNKENNESS

Instructions: (1) Clean the skin with soap and water and collect 5 ml. of blood from antecubital vein. Add 50 mg. of sodium fluoride and 15 mg. of potassium oxalate. The container should be tightly clamped and sealed. Rubber stoppers should be avoided. Refrigerate if sample cannot be sent immediately to the laboratory. (2) Opinion one is given if there is smell of alcohol in the breath, and physical examination reveals abnormal findings. (3) Opinion 2 is given if there is smell of alcohol in the breath, congested conjunctivae and dilated pupils, but other findings are normal. (4) Opinion 3 is given, if there is no smell of alcohol in the breath, and all the findings are normal. (5) The usual signs of drunkenness are: strong odour of alcohol in breath, loss of self-control and clearness of intellect, unsteady gait, vacant look, congested eyes, sluggish and dilated pupils, increased pulse rate, thick, slurred speech, talks at random, lack of perception of passage of time, and defective muscular coordination.

Appearances found on the person calling himself ..... age stated ..... years, an inhabitant of ..... who was sent with letter/ memo No. ..... dated ..... from ..... and accompanied by ..... for report as to the result of examination of person for certain appearances due to alleged drunkenness.

Preliminary Particulars:

(1) Name of the individual:
(2) Parent's or guardian's name:
(3) Address:
(4) Occupation:
(5) Caste:
(6) Sex:
(7) Age alleged by the person:
(8) Person accompanying or brought by:
(9) Consent (preferably in writing, with signature or left thumb impression):
(10) Name and number of accompanying P.C.:
(11) Identification marks: (1) (2)
(12) Date and place of examination:
(13) Time of commencement of examination:

History:

(1) Has he consumed alcohol? If so at what time and what was the nature and quantity?
(2) What food and drink he took and when?
(3) Is he in the habit of consuming alcohol regularly? If so, since how long and how frequently?
(4) Is he taking any medication or drugs? If so what is the nature and dose?
(5) If diabetic, when the last dose of insulin was taken and how much?

Physical Examination:

(1) B.P: Pulse: Temperature:
Weight: Height:
(2) General manners and behaviour (calm, talkative, abusive or obscene):
(3) State of dress (decent, soiled or disarranged):
(4) Speech (normal, thick, slurred, over-precise or incoherent):
(5) Self-control:
(6) Memory and mental alertness (memory of recent events, orientation of time and space):
(7) Handwriting (note time taken, repetition or omission of words, etc.):
(8) Pulse (rapid and bounding, slow, etc.):
(9) Temperature (surface temperature raised, lowered, normal):
(10) Skin: (dry, moist, flushed or pale):
(11) Mouth: (smell of alcohol, dribbling of saliva, furred tongue, dry lips, etc.):

(12) Eyes: (lids swollen or red, conjunctivae congested or not, visual acuity, pupils dilated or contracted; nystagmus (present or absent) and reaction of pupil to light and accommodation):
(13) Gait: (a) Manner of walking (unsteady/steady). (b) Reaction time to a direction to turn. (c) Manner of turning (normal or staggering):
(14) Stance: (Does he sway when standing erect with his feet together):
(15) Reflexes: (normal or depressed):
(16) Muscular co-ordination: (a) Placing finger to nose. (b) Placing finger to finger. (c) Unbuttoning and rebuttoning shirt. (d) Lighting cigarette with a match. (e) Walking along a straight line.
(17) Examination of C.V.S., R.S., G.I.S. and C.N.S. to rule out any disease.
(18) Laboratory investigations: Blood, Urine.

CERTIFICATE OF DRUNKENNESS

Signs of alcoholism found on the person of a male/female called (name) ..... aged ..... years, an inhabitant of ..... who was sent with letter/Cr. No. ..... dated..... from ..... of police, of P.S. ..... and accompanied by P.C. No. ..... for report as to his/her drunkenness.

Identification marks: (1) (2)

The person above noted was first seen by the undersigned at ..... A.M./P.M. on ..... and examination was commenced at ..... A.M./P.M. on ..... when the following signs were found.

Opinion: (1) Consumed alcohol and is under its influence (intoxicated) (2) Consumed alcohol, but is not under its influence. (3) Has not consumed alcohol.

Place: Signature:
Date: Name:
Designation:

PROFORMA FOR EXAMINATION OF SEXUAL OFFENCES
EXAMINATION OF THE VICTIM OF RAPE

Instructions: (1) Obtain written consent for examination and collection of specimens of blood, etc. and to supply copies of all medical reports to the police. (2) With cotton swabs take material from introitus, posterior fornix and cervical os and make four slides, and allow them to dry for two to three minutes. (3) Keep the same swab in test tube for acid phosphatase determination. (4) Insert a second swab in a test tube containing small amount of normal saline, and examine the fluid for motile spermatozoa. (5) Comb pubic hair and place loose hair in labelled envelope. (6) Cut sample of pubic hair (10 to 12 hairs) and place in labelled envelope. (7) Obtain blood 5 ml. (plain) for grouping and DNA profile, and 5 ml. (sodium fluoride) for alcohol and drugs and venereal disease. (8) Obtain fingernail scrapings and place in labelled envelopes. (9) Collect any loose hair or fibres found on the person or clothes. (10) Collect dried bloodstains and indicate the site from which collected. (11) Collect material from cervix for gonorrhoeal infection. (12) Obtain swabs from bite marks. (13) Preserve garments having suspected seminal or blood stains after drying them. (14) In most young women a finger may be passed into vagina although the hymen is intact. (15) If the vaginal opening is enough to admit two fingers easily and the hymen shows old tears, it indicates that the woman is used to sexual intercourse.

Appearances found on the person of female calling herself ..... age stated ..... years, an inhabitant of ..... who was sent with letter/memo No. ..... dated ..... from..... and accompanied by (name and number of P.C. of P.S.).....for report as to the result of examination of person for certain injuries or appearances said to have been caused on.....to be due to alleged rape.

Identification marks: (1) (2)

The person was seen by the undersigned on (date).....at (place of examination).....and the examination was commenced at.....A.M./P.M. on (date).....when the following were found.

Preliminary Particulars:

(1) Name of the individual:
(2) Parent's or guardian's name:
(3) Address:
(4) Occupation:
(5) Caste:
(6) Age as alleged by the victim:
(7) Person accompanying or brought by:

(8) Consent of the individual for examination:
(9) Signature of the individual consenting or her left thumb impression:
(10) In the case of minor, consent of the guardian and his/her signature or thumb impression:
(11) Married or single:
(12) Name of female attendant/nurse present at the time of examination:
(13) History as given by police:

History (From victim):

(1) Age:
(2) Gravidity:
(3) Age of menarche:
(4) Date of last menses:
(5) Whether pregnant:
(6) Most recent coitus prior to alleged assault:
Date: Time: Condom used or not:
(7) Patient's statement, whether she is a virgin:
(8) Is she suffering from any illness and taking any medicines:
(9) History of any venereal disease (past or present):
(10) History of emotional illness:
(11) Previous vaginal surgical procedure:
(12) Use of alcohol or drugs in twenty-four hours prior to alleged assault. If so amount and time of ingestion:
(13) (a) Date and time when the victim first made complaint to the police:
(b) Date and time when the rape was said to have been committed:
(c) Place where it was committed:
(d) Exact circumstances under which the rape was committed, i.e., whether the parties were standing or lying on the ground:
(e) Whether she was menstruating at the time:
(f) Whether she was sensible during the whole time the offence was committed or under the influence of alcohol or other intoxicants:
(g) Whether she uttered any cries or was she terrified:
(h) Did penis penetrate vulva?
(i) Did assailant wear condom?
(j) Since alleged assault has the victim:
(1) Douched (2) Bathed
(3) Defaecated (4) Urinated
(5) Changed clothes
(k) General feeling of those accompanying the female towards herself and towards the accused:

Physical Examination:

(1) B.P.: Pulse: Temp:
Wt: Height:
(2) General appearance:
(3) (a) Secondary sexual characters:
(b) Breasts: development, areola, nipple, milk in:
(4) Emotional status:
(5) Intelligence:
(6) Gait:
(7) Clothing worn at the time of alleged rape:
(a) Blood stains:
(b) Seminal stains:
(c) Other discharge:
(d) Foreign material:
(e) Hair: (f) Tears:
(g) Mud, dust, soil, grass stains, etc.:
(8) Body surface (injuries to breasts, cheeks, lips, thighs, genitals etc.):
(a) Scratches (b) Bruises
(c) Lacerations (d) Stains
(9) Mouth:
(10) Fingernails (damage, epithelial cells, foreign material, etc.):
(11) Genitals:
(a) Pubic hair, length, matted or not:
(b) Vulva (injuries and stains):
(c) Labia minora:
(d) Hymen (tears, recent or old):
(e) Vagina (digital and speculum exam):
(f) Fourchette (intact or torn):
(g) Perineum (tears, stains, scratches, foreign hair):
(h) Cervix:
(12) Is venereal disease present:
(13) Smears taken from vagina, cervix, urethra, sores:
(a) Number:
(b) Number of slides:
(c) Gram's stain:
(d) Stain for spermatozoa:
(14) Any other findings:
(15) Material sent to the laboratory:

(16) Results of laboratory procedures:
(a) Pregnancy test:
(b) Serology:
(c) Gonorrhoeal infection:
(d) Spermatozoa:
(e) Acid phosphatase:
(f) Grouping of blood and semen:
(g) Hair examination:

Collection of Evidence:

(1) Blood in two tubes for serology and grouping:
(2) Fingernail scrapings:
(3) Loose pubic hair:
(4) Cut pubic hair:
(5) Hair, fibres and blood stains on body and clothing:
(6) Vaginal material for live spermatozoa:
(7) Four slides from vaginal material:
(8) Cervical material for gonorrhoea:
(9) Vaginal secretion on two cotton swabs:
(a) Semen typing: (b) Acid phosphatase:

Opinion: (1) There are signs/no signs of recent vaginal penetration (if hymen is torn and external genitals show injuries). (2) There are signs of recent sexual intercourse (if semen is present in vagina). (3) There are signs of general physical injuries, and/or intoxication.

The signs are consistent/inconsistent with the history given.

Place: Signature:
Date: Name:
Designation:

EXAMINATION OF ACCUSED OF RAPE/SODOMY

Instructions: (1) Obtain written consent for examination, and collection of specimens of blood, etc. and to supply copies of all medical reports to the police. (2) Collect blood for comparison with semen type found in the victim. (3) In the case of accused in a rape case, swab from coronal sulcus, prepuce, penile shaft for blood comparison with the victim's blood. (4) With a cotton swab wipe glans penis, prepare two slides and dry for 2 to 3 minutes for vaginal and cervical cells. (5) Obtain penile washings for vaginal and cervical cells. (6) Look for smegma on corona glandis. It gets rubbed off during intercourse and usually requires about 24 hours to collect. (7) In the case of accused in a sodomy case, penis has peculiar smell of anal glands and may contain traces of lubricant and faecal matter. Fraenum may be torn and abrasion seen on prepuce and glans. Faecal soiling, blood and foreign hairs may be found on coronal sulcus. (8) In the case of buccal coitus, faint teeth marks and abrasions may be seen on penis. (9) Urethral discharge for gonococcal infection. (10) Comb pubic hair and place loose hair in an envelope. (11) Obtain fingernail scrapings and place in labelled envelope. (12) Preserve garments having suspected seminal stains.

Preliminary particulars and reference, etc.:

As in the case of the victim.

History:

(1) Brief description of assault: Record general history and omit history of specific incident. Find out how injuries found on body were caused.
(2) Date and time of assault:
(3) Is he suffering from any illness and taking any medicines?
(4) Use of alcohol or drugs in 24 hours prior to alleged assault, if so amount and time of ingestion:
(5) Past illness, surgical operations, serious accidents:
(6) Consenting intercourse with any woman within previous 24 hours:

Physical Examination:

(1) B.P.: Pulse: Temp:
Wt: Height: General build:
(2) General appearance and emotional status:
(3) Clothing (changed or not):
(a) Blood stains:
(b) Seminal stains:
(c) Foreign material:
(d) Hairs:
(e) Tears:
(f) Cosmetic contact traces:
(g) Mud, soil, grass, etc:
(4) Fingernails (damage, epithelial cells, foreign material, etc.):
(5) Influence of alcohol or other intoxicant:
(6) Injuries found on body (scratches, bruises, bite marks, etc.):
(7) Genitals (development with special reference to potency):
(a) Pubic hair: length, matted or not:
(b) Penis: Length when flaccid:

Length when erect:
(c) Injuries: (d) Blood, semen, lubricant, faecal matter:
(e) Smegma:
(f) Prepuce (abrasions, female pubic hair):
(g) Coronal sulcus (faecal soiling, blood and pubic hair):
(h) Circumcised or not:
(i) Fraenum (tears):
(j) Scrotum and testis (development, injuries):

(8) Anal examination:
(9) During alleged assault:
Did penis penetrate vagina/rectum?
Did assailant had orgasm?
Did assailant wear condom?
(10) Since alleged assault:
Bathed: Urinated:
Defaecated:
(11) Is venereal disease present?
(12) Material sent to laboratory:
(13) Results of laboratory procedures:
(14) Any other finding:

Collection of Evidence:

(1) Blood in two tubes for serology and grouping and for drug screening. (2) Perianal swab. (3) Rectal swab. (4) Oral swab. (5) Loose pubic hair. (6) Hair, fibres and blood stains on body and clothing. (7) Urethral discharge for gonorrhoea. (8) Fingernail scrapings. (9) Suspected dried seminal stains on the skin or clothing. (10) Penile washings (presence of vaginal epithelium).

Opinion: (1) There are no signs indicative of recent vaginal/anal penetration. (2) There are signs indicative of recent vaginal epithelial cells, lubricant or faecal stains on penis.

EXAMINATION OF THE VICTIM OF SODOMY

Instructions: (1) Obtain written consent for examination, and collection of specimens of blood, etc. and to supply copies of all medical reports to the police. (2) With a cotton swab wipe the perianal area, and put in a test tube for acid phosphatase determination. (3) Insert a cotton swab into the anal canal without touching the perianal area. Prepare two slides and dry for 2 to 3 minutes. (4) Place the same swab in a tube containing normal saline for acid phosphatase determination. (5) Insert a second swab in a test tube containing small amount of normal saline, and examine the fluid for motile spermatozoa. (6) Cut sample of pubic hair (10 to 12 hairs) and place in labelled envelope. (7) Obtain blood samples (two) for comparison with grouping of the semen and for serological test. (8) Obtain fingernail scrapings and place in labelled envelopes. (9) Collect any loose hair or fibres found on the person or clothes. (10) Collect dry blood stains and indicate the site from which collected. (11) Collect material from anus for gonorrhoeal infection. (12) In buccal coitus, swab the mouth of the patient, especially the gums and pharynx with a cotton swab, smear on a slide and dry. (13) Place the same cotton swab in a tube containing normal saline for acid phosphatase.

Appearances found on the person of a male/female calling himself/herself ..... age stated ..... years, an inhabitant of ..... who was sent with letter/memo no. ..... dated ..... from ..... and accompanied by ..... for report as to result of examination of person for certain injuries or appearances said to have been caused on .....to be due to alleged sodomy.

Identification marks: (1) ..... (2) .....

The person was seen by the undersigned on (date and time) .....and the examination was commenced at (date and time) .....when the following were found:

(1) Name of the individual:
(2) Parent's or guardian's name:
(3) Address:
(4) Occupation:
(5) Caste:
(6) Age as alleged by the victim:
(7) Persons accompanying or brought by:
(8) Consent of the individual for examination:
(9) Signature of the individual consenting or his/her left thumb impression:
(10) In the case of minor, consent of the guardian and his/her signature or thumb impression:
(11) Name of the female attendant/nurse, present at the time of examination:

History (from victim)

(1) Is he/she suffering from any illness and taking any medicines:

(2) History of any venereal disease (past or present):
(3) History of emotional illness:
(4) Use of alcohol or drugs in 24 hours prior to alleged assault. If so amount and time of ingestion:
(5) Date and time when the victim first made complaint to the police:
(6) Date and time when sodomy was said to have been committed:
(7) Place where it was committed:
(8) Exact circumstances under which sodomy was committed, i.e., whether the parties were standing or lying on the ground:
(9) Whether he/she uttered any cries or was terrified:
(10) Whether he/she was sensible during the whole time the offence was committed or under the influence of alcohol or other intoxicants:
(11) Details of struggle or resistance:
(12) Use of any lubricant:
(13) Did penis penetrate anus? (degree of penetration):
(14) Did assailant wear condom?
(15) Ejaculation during the act:
(16) Pain experienced at the time of act or subsequently:
(17) Any bleeding:
(18) Since alleged assault has the victim:
(a) Bathed or washed the anal area:
(b) Defaecated:
(c) Urinated:
(d) Changed clothes:

Physical Examination:

(1) B.P.: Pulse: Temp:
Wt: Height: Build:
(2) General appearance:
(3) Emotional status:
(4) Clothing worn at the time of offence: (a) Blood stains. (b) Seminal stains. (c) Faecal stains. (d) Mud, dust, soil, grass stains, etc. (e) Foreign material. (f) Hair. (g) Tears.
(5) Marks of violence on the body (scratches, bruises, bite-marks, etc.):
(6) Any type of stains or foreign bodies on the body:
(7) Fingernails (damage, epithelial cells, foreign material, etc.):
(8) Mouth (in buccal coitus):
(9) Anal examination: (a) Inspection: Look for recent tears, stains due to semen, blood, faecal matter around the anus, scars, piles, condyloma, shaving of anal hair, smoothness or thickness of anal mucosa, depression of buttocks towards anus, etc. (b) Digital examination (assess tone and degree of dilatation of sphincter, discomfort or pain during examination). (c) Lateral buttock traction test. (d) Speculum examination (look for abrasions, contusions, lacerations, piles, fissures, wrinkles in anal mucosa, etc.):
(10) Any other findings:
(11) **Specimens to be collected:** (1) Blood. (2) Head hair. (3) Pubic hair. (4) Loose hair and fibres found anywhere on the body. (5) Swabs from any soiled areas of the skin. (6) Swabs from the anal and lower rectum. (7) Nail scrapings.
(12) Results of laboratory procedures:

Opinion: (1) Findings are not consistent with entry of a penis into anus. (2) Findings are consistent with entry of a penis into anus. (3) Sodomy has been committed (if semen is present in the anus):

Place: Signature:
Date: Name:
Designation:

PROFORMA FOR EXAMINATION OF CASE OF IMPOTENCY/POTENCY

Appearances found on the person of a male/female calling himself/herself ..... age stated ..... years, an inhabitant of ..... who was sent with letter/memo no ..... dated ..... from ..... of police of P.S. ..... and accompanied by (name and No. of P.C. ..... of P.S. .... for report as to the result of examination for potency/impotency.

(1) Name of the individual:
(2) Age:
(3) Sex:
(4) Address:
(5) Occupation:
(6) Brought by:
(7) Date, time and place of examination:
(8) Consent of the individual for examination: (Signature or left thumb impression):
(9) Marks of identification: (1) (2)

(10) **History of the case:** (Diabetes, drug addiction, trauma, STD, etc., masturbation, night emissions, homosexual/sexual intercourse):
(11) **General examination:**
Height: Weight:
Secondary sexual characters:
Physique:
(12) **Local examination:**
(a) Penis: Length when flaccid:
Length when erect:
Circumference (flaccid and erect):
Disease/deformity/injury (if any):
Sensation over glans penis:
Foreskin: Retractable/non-retractable:
(b) Scrotum (pendulous or not):
(c) Testis: Whether present in scrotum or not; size (small, medium, adult size):
(13) **Systemic examination:**
(a) C.V.S.
(b) R.S.
(c) G.I.S.
(d) G.U.S.
(e) C.N.S.
(14) (a) Urine (for sugar):
(b) Blood/VDRL:
(c) Others:

Place: Signature:
Date: Name:
Designation:

CERTIFICATE OF POTENCY/ IMPOTENCY OF MALE

Certified that I examined Mr ..... on (date to be given) at (place of examination to be given) and found as follows:

(1) That his genital organs are normal/not of normal development:
(2) That there is evidence/no evidence of disease of testes or epididymides:
(3) That he gives no history/definite history of exposure and had venereal disease:
(4) That there is no evidence of organic disease of nervous system/that his nervous system shows evidence of organic disease:
(5) That there are well-marked secondary sex characters/secondary sex characters are not marked as they ought to be in a man of his age.
(6) That there are psychic causes/no psychic causes:

Opinion: From all (or some) of the aforementioned objective signs, it can be concluded that (1) There is nothing to suggest that the male examined is not capable of performing sexual act. (2) The person is incapable of performing sexual act.

Place: Signature:
Date: Name:
Designation:

CERTIFICATE OF POTENCY/ IMPOTENCY OF FEMALE

Certified that I examined Mrs. ..... on (date) at (place) and observed as follows:

(1) That the labia majora and minora were well-developed/ill-developed.
(2) That she has normal vagina/total absence of the vagina, or had a (short) (narrow) vagina with a very small, ill-defined opening.
(3) That her hymen was thick and of a rigid type, not even admitting a small finger, intact/ruptured.
(4) That, at the time of examination, though her genitals were of normal development with an elastic hymen, she complained of excruciating pain and screamed when the inner aspect of the vulva was touched/no vaginismus.
(5) She has/has no apparent discharge or venereal diseases.
(6) That there are psychic causes/no psychic causes.

Opinion: From the above observations: (1) I am of the opinion that she is incapable of taking part in sexual intercourse (impotent), i.e., she is not apta vira and that she is still a virgin. (2) She is apta vira, i.e., capable of taking part in sexual intercourse.

Place: Signature:
Date: Name:
Designation:

HEALTH AND AGE CERTIFICATE

I hereby certify that I have examined Sri ....a candidate for employment in .....office/ department, and cannot discover that he/she has any disease (communicable or otherwise), constitutional weakness or bodily infirmity, except .....

I do not consider this a disqualification for employment in the office of the ..... Sri ..... age is, according to his/her own

statement ..... years, and by appearance about ..... years.

Signature of the candidate: Signature:
Station: Name:
Date: Designation:
Reg. No.:
State medical council of ..... State

BIRTH CERTIFICATE

Name: Sex:
Date of birth:
Place of birth:
Name of father:
Name of mother:
Residential address:
Date:
Seal: Signature of doctor:
Name:
Registration. No.
State medical council of ..... State

MEDICAL CERTIFICATE RECOMMENDED FOR LEAVE OR EXTENSION OF LEAVE

Signature of applicant or L.T.I.
Identification Marks: (1) (2)
To be filled in by the applicant in the presence of Govt. doctor/medical practitioner.

I, Dr. ..... after careful examination of the case hereby certify that (name of patient) ..... aged ..... sex ..... resident of ....., whose signature is given above is suffering from ..... and I consider that a period of absence from duty of ..... days with effect from ..... is absolutely necessary for the restoration of his/her health.

Place: Signature, Name and Stamp of Dr.
Date: Reg. No.:
Time: State medical council of .....State

MEDICAL CERTIFICATE OF FITNESS TO RETURN TO DUTY

Signature of applicant of L.T.I. _____
Identification Marks: (1) (2)
To be filled in by the applicant in the presence of Govt. doctor/medical practitioner.

I, Dr. ..... do hereby certify that I have carefully examined ..... of the department ..... whose signature is given above and find that he has recovered from his illness ..... with which he was suffering since ..... and is now fit to resume his duties in Govt. services w.e.f. ..... I also certify that before arriving at this decision, I have examined the original medical certificates and statement of the case (or certified copies thereof) on which leave was granted or extended and have taken these into consideration in arriving at my decision.

Place: Signature, name and stamp of Dr.
Date: Reg. No.
Time: State medical council of ..... State

ANNEXURE 1 (A)

FORM NO. 4

MEDICAL CERTIFICATE OF CAUSE OF DEATH

(Hospital in-patients. Not to be used for stillbirths)

To be sent to Registrar along with Form No. 2 (Death Report)

Name of the Hospital .....

I hereby certify that person whose particulars are given below died in the hospital in Ward No. ..... on ..... at ..... A.M./P.M.

<table>
<tr><td colspan="4">Cr. No. ..... Date of Admission .....
Name of Deceased .....S/W/D/of .....
..... Address .....
.....</td><td>For use of Statistical Office</td></tr>
<tr><td rowspan="2">Sex</td><td colspan="3">Age at Death</td><td rowspan="3"></td></tr>
<tr><td>If 1 year or more, age in years</td><td>If less than 1 year, age in months</td><td>If less than 1 month, age in days</td><td>If less than 1 day age in hours</td></tr>
<tr><td>1. Male</td><td></td><td></td><td></td><td></td></tr>
<tr><td>2. Female</td><td></td><td></td><td></td><td></td><td></td></tr>
<tr><td colspan="3">Cause of Death</td><td>Interval between onset and death approx.</td><td></td></tr>
<tr><td colspan="2">I. Immediate cause
State the disease, injury or complication which caused death, not the mode of dying, such as heart failure, asthenia, etc.
Antecedent cause
Morbid conditions, if any, giving rise to the above cause, stating underlying conditions last.
II. Other significant condition contributing to the death but not related to the disease or conditions causing it.</td><td>(a) .....
due to (or as a consequences of
(b) .....
due to (or as a consequences of
(c) .....
.....
.....
.....
.....</td><td>.....

.....

.....
.....
.....
.....</td><td>.....

.....

.....
.....
.....
.....</td></tr>
<tr><td colspan="2">Manner of Death
(1) Natural. (2) Accident. (3) Suicide.
(4) Homicide. (5) Pending investigation.</td><td colspan="3">How did the injury occur?</td></tr>
<tr><td colspan="3">If deceased was a female, was pregnancy the death associated with?
If yes, was there a delivery?</td><td colspan="2">(1) Yes (2) No
(1) Yes (2) No</td></tr>
</table>

Name and signature of the Medical Attendant certifying the cause of death

Date of verification .....

(To be detached and handed over to the relative of the deceased)

Certified that Shri/Smt./Kum. ..... S/W/D/ of Shri ..... CR. No. ..... R/o ..... was admitted to this hospital in ward ..... on ..... and expired on .....

Doctor's Signature

(Medical Supdt. Name of the Hospital)

Weights and measurements of organs in adults

Brain: 1,400 g (males); 1275 g (females); 1.4% body weight.
Spinal cord: 27 to 28 g; length 45 cm.
Pituitary: 0.5 to 0.6 g; size 2.1 × 1.4 × 0.5 cm.
Heart: 300 g (males); 250 g. (females); 0.4 to 0.45% body weight. Length, 12.5 cm; Width 9 to 10 cm. (at atrioventricular groove). Circumference: 24 to 25 cm.
Thickness of walls of atria: 1 to 2 mm.
Thickness of walls of right ventricle: 3 to 5 mm.
Thickness of walls of left ventricle: 10 to 15 mm.
Circumference of aortic valve: 7.5 cm. (6 to 7.5 cm.).
Circumference of pulmonary valve: 8.5 cm. (7 to 9 cm.).
Circumference of mitral valve: 10 cm. (8 to 10.5 cm.)
Circumference of tricuspid valve: 12 cm. (10 to 12.5 cm.).
Thyroid: 20 to 40 g.; size 5 to 7 × 3 to 4 × 1.5 to 2.5 cm.
Parathyroids: 0.12 to 0.18 g; Thymus 15 to 40 g.
Right lung: 360 to 570 g; mean 450 g. Both lungs 1% of body weight.
Left lung: 325 to 480 g.; mean 375 g.
Trachea: 11 to 12 cm.; **Right bronchus** 2.5 cm.; **Left** 3.5 to 5 cm.
Liver: 1400 to 1500 g.; 1.8% of body weight.
Spleen: 150 to 200 g; 0.16% of body weight.
Kidney: 130 to 160 g. (males); 120 to 150 g. (females); size: 11 to 12 × 5 to 6 × 3 to 4 cm.
Pancreas: 90 to 120 g; size: 23 cm × 4.5 cm × 3.8 cm; 0.1% body weight.
Testis: 20 to 27 g.; size: 4 to 5 × 2.5 to 3.5 × 2 to 2.7 cm.
Prostate: 15 to 20 g; size: 3.6 × 2.8 × 1.9 cm.
Uterus: Virgin: 30 to 40 g; size: 7.8 to 8 × 3.4 to 4.5 × 1.8 to 2.7 cm.
Parous: 100 to 120 g; 8.7 to 9.4 × 5.4 to 6.1 × 3.2 to 3.6 cm.
Ovary: 7 g; size: 2.7 to 4.1 × 1.5 to 0.8 cm.
Suprarenals: 5 to 6 g.
Pharynx: 11 to 12 cm; **oesophagus:** length 25 cm.
Stomach: length 25 to 30 cm. weight: 300 g; capacity 1100 to 1200 ml.
Small intestine: length 550 to 650 cm; weight 800 to 900 g.
Duodenum: 25 to 30 cm: **Jejunum** (upper 2/5:) **Ileum** (lower 3/5).
Bile ducts: 7.5 cm.
Large intestine: length 150 to 170 cm; weight: 600 g; **Appendix** 7.5 to 10 cm.
Caecum: 6 to 7 cm long; Ascending colon, 20 cm; Transverse colon 50 cm.
Descending colon: 12 to 15 cm; Pelvic colon, 40 to 45 cm; Rectum 12 to 15 cm.
Anal canal: 2.5 to 4 cm.
Inguinal canal: 3.5 cm.
Thoracic duct: 40 to 45 cm.
Bladder capacity: 250 ml; Ureter: 25 cm.
Male urethra: 20 to 25 cm; prostatic 2.5 to 4 cm; membranous 2 cm; spongy and penile 15 to 20 cm.
Female urethra: 2.5 to 4 cm.
Vas deferens: 40 to 45 cm.
Uterine tubes: 10 to 12 cm.
Vagina: anterior wall 6 cm; posterior wall 9 cm.
Placenta: 500 g; size: 16 to 20 × 2.5 to 3 cm.
Bones: 11.6% of body weight.
Skeletal muscle: 28.7% of body weight.
Fat: Males 20 to 25%; **Females** 25 to 30% of body weight.
The child's brain attains mature size and weight at about 6 years of age.

THE SYNOPSIS OF
FORENSIC MEDICINE
AND
TOXICOLOGY

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